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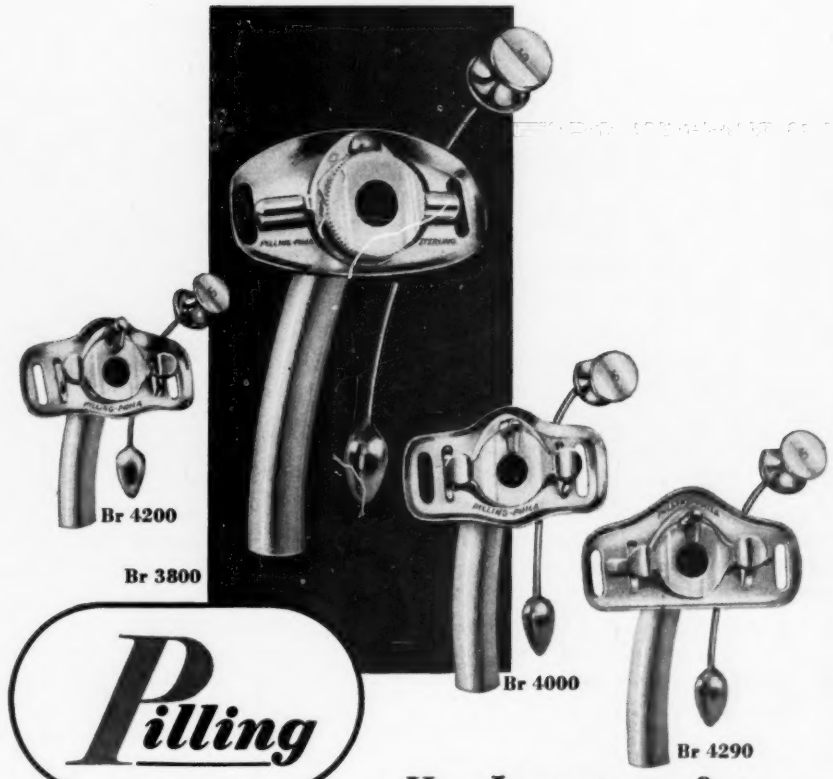

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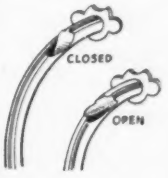

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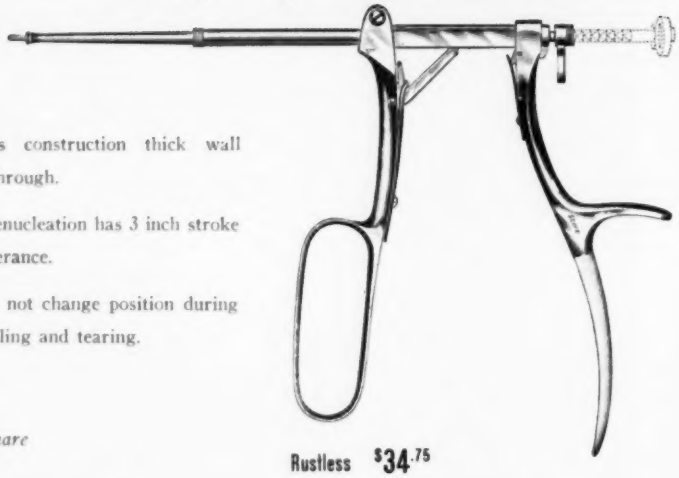

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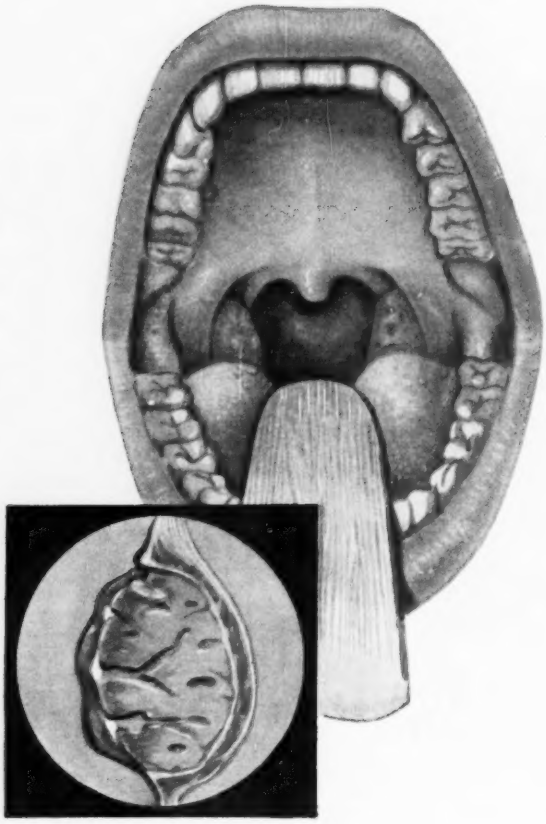

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VOLUME 55 JANUARY 1952 NUMBER 1

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CALCIFICATION IN THE LARYNGEAL CARTILAGES

JACOB H. VASTINE II, M.D.
AND
MARY FRANCES VASTINE, M.D.
PHILADELPHIA

THE PATTERN of calcification in the laryngeal cartilages is similar in identical twins. This fact would seem to indicate that the deposition of calcium in these areas is hereditarily determined. Therefore, acquired factors such as disease or disturbances in the mineral metabolism probably have very little, if anything, to do with this phenomenon. This conclusion has been reached because in the course of a study of the calcification in the costal cartilages of identical twins [1] it occurred to us that a similar study of the laryngeal cartilages of identical twins might prove interesting. Accordingly, lateral films of the neck were obtained in five pairs of identical twins. The roentgen findings in each pair are described.

The calcification seen posteriorly in the thyroid cartilages of J. S. and M. S., identical 20-year-old twins, is seen to be strikingly similar in pattern and distribution (Fig. 2). It is notable, too, how similar are the contours of the hyoid bones in these young women. It is also true that their cervical spines are so nearly alike that they could almost be superimposed one on the other.

In the second pair of twins, A. B. and J. B., 64-year-old men, the laminae of the thyroid cartilages are calcified in their entirety, and the calcified inferior cornua are visualized through the calcified laminae of the cricoid cartilages (Fig. 4). This widespread calcification enables one to visualize them and to realize how much alike these structures really are. It is also quite evident from these films that the degenerative changes in the cervical spine, although present in both of the twins, are not the same.

Figure 5 presents identical twins, Mrs. E. B. and Mrs. E. S. Here again the pattern of calcification in the thyroid laminae of Mrs. E. B. is quite different from that seen in any other pair of twins studied but plainly similar to that of her twin sister Mrs. E. S. (Fig. 6). The superior cornua are well visualized in these women because of the ossification. The cricoid laminae are also well seen for the same reason. In these twins, unlike A. B. and J. B., who were discussed previously (Fig. 4), the degenerative changes in the cervical spines are very much alike.

1. Vastine, J. H., II; Vastine, M. F., and Arango, O.: Genetic Influence on Osseous Development with Particular Reference to the Deposition of Calcium in the Costal Cartilages, Am. J. Roentgenol. **59**:213-221 (Feb.) 1948.

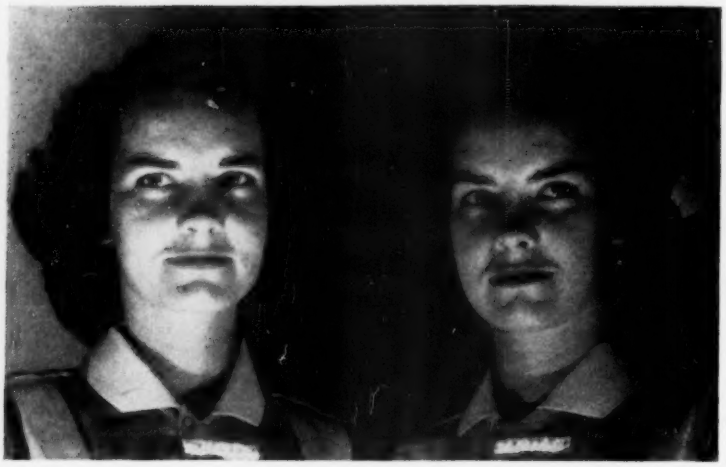

Fig. 1.—Identical twins J. S. and M. S.

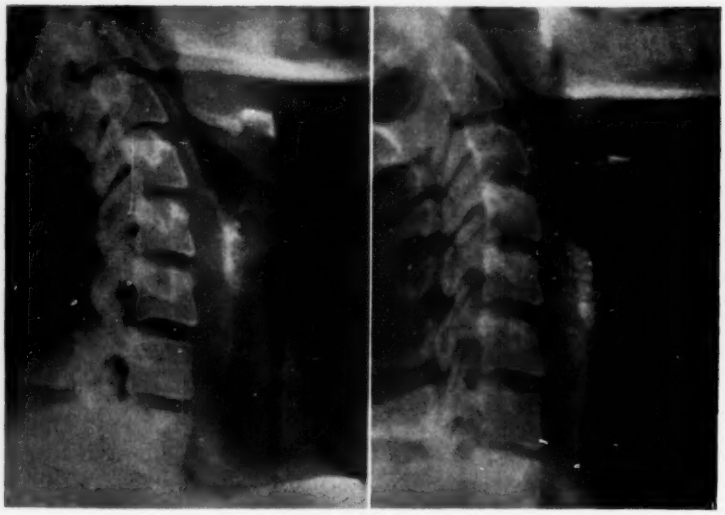

Fig. 2.—Roentgenograms of twins J. S. and M. S. The calcification in the thyroid cartilages of these 20-year-old twins is strikingly similar.

Fig. 3.—Identical twins A. B. and J. B.

Fig. 4.—Roentgenograms of twins A. B. and J. B. The widespread calcification in the thyroid cartilages of these 64-year-old twins enables one to realize how much alike these structures really are.

Fig. 5.—Identical twins Mrs. E. B. and Mrs. E. S.

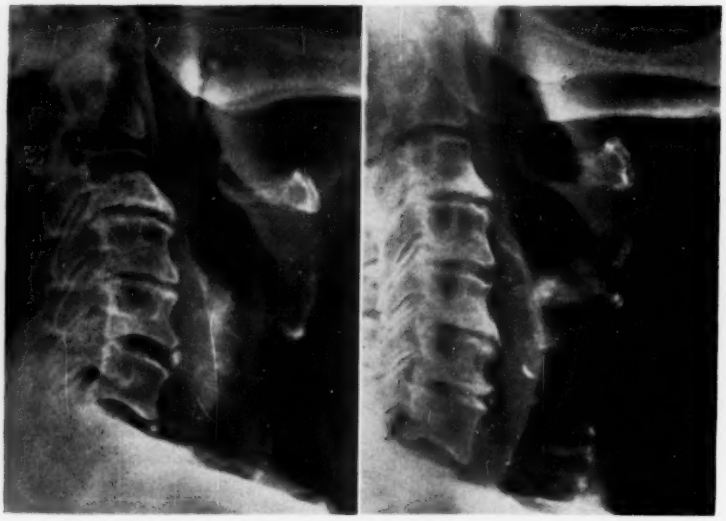

Fig. 6.—Roentgenograms of Mrs. E. B. and Mrs. E. S. The superior cornua of the thyroid cartilages and the cricoid laminae are well visualized in these twins. The degenerative changes in their cervical spines are much alike.

Fig. 7.—Identical twins R. B. and W. B.

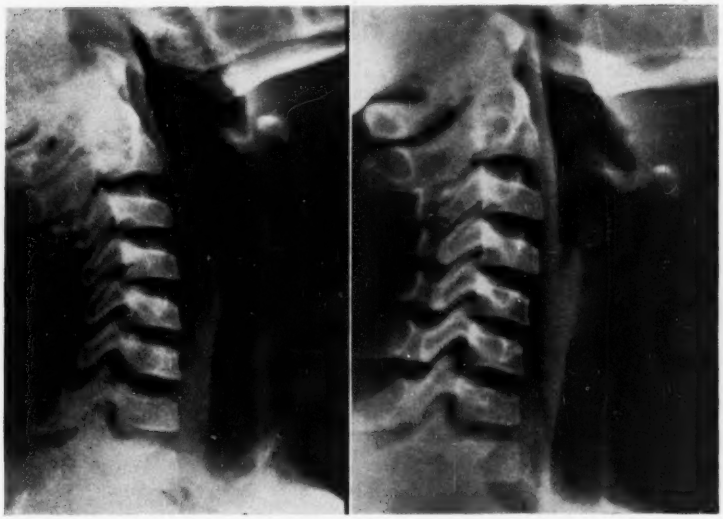

Fig. 8.—Roentgenograms of twins R. B. and W. B. These 13-year-old boys show no calcium in their laryngeal cartilages as yet. However, their hyoid bones and cervical spines are almost identical.

Fig. 9.—Identical twins M. A. and N. A.

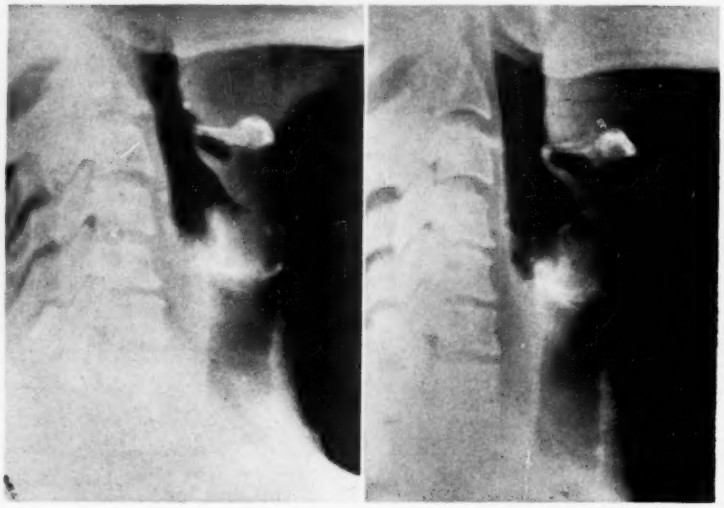

Fig. 10.—Roentgenograms of twins M. A. and N. A. Note the similar rims of calcification in the thyroid cartilages of these twins; also the small round calcifications just back of the anterior marginal rims. The small corniculate cartilages are well visualized.

R. B. and W. B., 13-year-old identical twins (Fig. 7), show no calcium in their laryngeal cartilages as yet. However, the contour of the hyoid bones and of the cervical spines (Fig. 8) is almost identical.

The rim of calcification seen anteriorly and superiorly in the thyroid cartilages of both M. A. and N. A., identical twins (Fig. 9), is interesting. In both, there is a small round area of calcification just posterior to the anterior marginal rim of ossification (Fig. 10). The small corniculate cartilages are well seen perched on the tips of the apparently identical superior cornua of the thyroid cartilages. Also notable is the striking similarity in the cricoid laminae. In both N. A. and M. A., the lesser cornua of the hyoid bones are well seen.

In a previous publication [1] the fact was brought out that the size, shape, and contour of the skeletal system is predetermined. Even the most minute detail of ossification is under the direct influence of the genes. This is evident when a close comparison of the roentgenograms of each of the respective bones of identical twins is made. If the detail of the cervical spines of each twin in the accompanying illustrations is studied, the close similarity in the bony make-up of the respective twin pairs is evident. In fact, as we have suggested,[1] this amazing similarity in the bony structure may prove to be our most accurate criterion for differentiating homozygotic (developed from one fertilized egg) from dizygotic (developed from two fertilized eggs) twins. Of course, degenerative changes or other conditions acquired after birth may obscure the original picture. Thus, in Figure 4, the prominent osteoarthritic changes in the one brother and the absence of such degenerative changes in his twin cause these spines to present a rather different appearance. On the other hand, the degenerative changes exemplified in Figure 6 occur in cervical spines which remain very similar even after 70 years of postnatal life.

SUMMARY

These studies of 10 patients constituting five pairs of identical twins have been presented in some detail to bring out the following points:

1. The deposition of calcium in the laryngeal cartilages is strikingly similar in identical twins.

2. It is reasonable to assume, therefore, that the deposition of calcium in the laryngeal cartilages is hereditarily determined and not influenced by or due to any acquired factor, such as disease, mineral deficiency, or metabolic disturbance.

3. The configuration of the hyoid bones and of the cervical spines is also similar in identical twins.

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HEMORRHAGE IN THE NOSE AND THROAT

HERBERT P. HARKINS, M.D., M.Sc.(Med.)
PHILADELPHIA

BLEEDING is the commonest surgical complication in the field of otolaryngology. Therefore, its recognition and prompt control must be mastered by every ear, nose, and throat practitioner. This is not always easy, particularly if one is not observant and alert, but with reasonable care and diligence there is little reason to be troubled. However, if we place too great or too prolonged dependence on simple measures for control, disaster will sooner or later overtake us. All otolaryngologists have troublesome postoperative hemorrhages from both tonsillectomies and adenoidectomies. Spontaneous nasal hemorrhage in arteriosclerotic and hypertensive patients is very common, and trauma to the nose and face with resultant troublesome bleeding is seen almost daily. These, plus hemorrhage following nasal surgery and bleeding due to tumors in the nose, pharynx, and larynx, constitute the bulk of these complications and consequently deserve careful attention.

The following general facts concerning hemorrhage should be borne in mind: 1. Bleeding in any quantity is always attended by a proportionate amount of surgical shock. 2. In children, shock often appears from relatively small blood losses, while in an adult, very large amounts may drain away before any alarming symptoms are noticed. 3. In a bleeding adult, one is notified of any hemorrhage quickly, for the patient himself is on the alert and recognizes the importance of blood loss; whereas in children, bleeding is often insidious, and the young patient may drift into a stage of dangerous anoxemia or anoxia while neither he nor anyone around him is aware of what is happening. 4. The common symptoms of hemorrhage are increasing pulse rate, drop in blood pressure, restlessness, anxiety, desire for fluids, increasing pallor, and later, air hunger.

These facts should be remembered in nose and throat bleeding: 1. Postoperative nasal bleeding in children usually indicates that the blood is coming from the adenoid area of the nasopharynx. 2. In postoperative tonsil bleeding, the blood is expectorated first, and later may appear in the nose. 3. Severe nasal hemorrhage occurs usually high up and far back in the nose and usually comes from the sphenopalatine or the nasopalatine arteries or veins. It is accompanied by a profuse flow of blood into the pharynx. 4. Bleeding from Kieselbach's plexus or Little's area on the septum, while alarming at times, is usually easily controlled by pressure, and rarely results in much blood running into the throat.

Even in the presence of these symptoms, it is sometimes difficult to impart to an intern or resident the significance of nasal or pharyngeal bleeding. Often it is considered normal for a certain amount of hemorrhage to occur after nose or

Presented before the Philadelphia Laryngological Society, March, 1951.

Submitted for publication Sept. 4, 1951.

throat surgery, whereas the same amount of bleeding following abdominal or urologic surgery would quickly arouse the staff to the most heroic measures. This attitude can be corrected only by the staff physician's insistence and example.

As all otolaryngologists know, most postoperative tonsillectomy and adenoidectomy bleeding occurs during the first 12 hours following surgery. Hence, the patient should be most carefully watched during this period. To fail to provide adequate care is to tempt fate and court trouble. During this time, the patient must be observed for increased swallowing, restlessness, anxiety, expectoration of bright red blood, and increase in pulse rate. Any of these symptoms calls for immediate and careful examination of the nose, nasopharynx, and oropharynx to locate any bleeding areas. All clots seen in the tonsil fossae and adenoid areas should be promptly removed, as bleeding behind them is common and insidious. All structures should be examined, regardless of the resistance of the patient. One should never walk away contented or satisfied until all possible bleeding areas have been completely explored.

When confronted with a child who has obviously been bleeding for a considerable time and may be bordering on or actually in surgical shock, one should never allow an inhalation anesthetic to be given until an adequate transfusion of whole blood has been given. If immediate suturing is required in such cases, it must be done under local infiltration of procaine hydrochloride, or with no anesthesia. Inhalation anesthetics may produce rapid anoxia and death in an exsanguinated child.

If a slight ooze is seen in one or both tonsil fossae after clot removal, the topical application of thrombin in powder or liquid form on a tampon may control it. Tannic and gallic acid, in equal parts, with epinephrine 1 : 1,000 added, may also be tried on a gauze sponge. The submucosal infiltration of 1% procaine hydrochloride with or without epinephrine 1 : 1,000, may also be attempted. If, however, the bleeding area is not dry in five to eight minutes, it is useless to continue, and the area should be closed by ligature or suture. Should there be extensive hemorrhage, a tampon of oxidized cellulose may be placed tightly in the fossa and held there until the anterior and posterior pillars are sutured together over it. It is, of course, unnecessary to remove either the sutures or the pack. This method will stop almost any bleeding in a tonsil fossa.

Fortier [1] believes that ligatures are satisfactory for controlling postoperative fossa bleeding, especially if they are secured with the La Force tier. With this opinion I cannot wholly agree. The slipping of a ligature on one occasion, with resultant serious bleeding, has made me reluctant to rely on this method. If the ligature remained still, it might be dependable, as it is in general surgical procedures. However, with the pharyngeal musculature in almost constant motion, any ligature can easily slip off, with disastrous results. For the postoperative control of most nasopharyngeal or adenoid bleeding, only the postnasal tampon is truly dependable, unless one is able satisfactorily to visualize and suture the bleeding point. Suturing is difficult in the nasopharynx because of limited exposure. Ligatures here are troublesome to use for the same reason.

1. Fortier, G. M. A.: Hemostasis in Tonsillectomy, Minnesota Med. **31**:493-496 (May) 1948.

A properly placed postnasal tampon, with tension maintained, will promptly stop almost all bleeding in this area. The value of the pack is further enhanced by inserting tight-fitting anterior nasal dressings of oxidized cellulose. These dressings should occupy the entire nasal chamber on both sides and extend posteriorly to meet the nasopharyngeal tampon. This combination will allow the otolaryngologist to remain confident in the face of almost any nasopharyngeal or posterior nasal bleeding. Houser [2] and Kler [3] have both found oxidized cellulose to be effective, nonirritating, and safe for the satisfactory control of spontaneous nasal hemorrhage, as well as bleeding following intranasal and nasopharyngeal surgery.

Postnasal packs should never remain in place longer than 48 hours, because of the danger of inciting Eustachian tube or middle ear disease. If bleeding recurs after their removal, they may be reinserted later or another method of control employed.

When hemorrhage of the nose and throat is not satisfactorily controlled by one of these methods, or a modification, then ligation of the external carotid artery should be considered. This ligation should be done above the superior thyroid artery at the level of or slightly above the superior cornu of the thyroid cartilage. External carotid ligations are not dangerous, if one is sure to ligate the correct vessel. The artery in the carotid sheath with branches in the neck is the proper one.

Of 106 patients admitted to the Mayo Clinic [4] from 1930 to 1947 for severe epistaxis, there were 11 who required ligation of the external carotid artery. All bleeding was effectively controlled by the operation. Two of my patients required external carotid ligation, of a total of 32 patients with severe epistaxis. After ligation, bleeding promptly stopped in both.

PREVENTIVE MEASURES

Recently much investigative work has been done on the causes of postoperative hemorrhage, especially of delayed bleeding. Hypovitaminosis K, hypovitaminosis C, and hypoprothrombinemia resulting from acetylsalicylic acid and other salicylate therapy have been carefully studied. Fox and West,[5] at the University of Maryland, in a study of 687 adenotonsillectomy patients, found that vitamin K afforded very little protection in late tonsillar hemorrhage. In a group of 128 patients who received 10 mg. of vitamin K twice daily by mouth, 10.1%, or 13 patients, had delayed bleeding. In 216 patients who received only salicylic acid chewing gum, there were 21 patients, or 9.7%, who exhibited late hemorrhage. The remaining 343 patients received no vitamin therapy and no acetylsalicylic acid or salicylic acid chewing gum, and there were only two instances of hemorrhage among them, or a percentage of 0.58. Of this group, all over 12 years of age (159 patients)

2. Houser, K. M.: Oxidized Cellulose Gauze Packing for Nasal Bleeding, J. A. M. A. **132**:143 (Sept. 21) 1946.

3. Kler, J. H.: Use of Absorbable Gauze in Otolaryngologic Practice, Arch. Otolaryng. **46**:801-803 (Dec.) 1947.

4. Spar, A. A., and Hallberg, O. E.: Severe Epistaxis and Its Management: Report of 11 Cases in Which External Carotid Artery Was Ligated, Ann. Otol. Rhin. & Laryng. **56**:141-151 (March) 1947.

5. Fox, S. L., and West, G. B., Jr.: Vitamin K and Late Tonsillar Hemorrhage, Bull. School Med. Univ. Maryland **32**:215-220 (April) 1948.

received troches containing ethyl aminobenzoate, benzocaine, and tyrothricin as desired for discomfort. The very low incidence of late bleeding in this group is believed by the observers to be due to a cleaner fossa, free from secondary infection and excessive granulation tissue. It is their conclusion, too, that the increased number of secondary bleeders in the first two groups was the result of secondary infection of the fossae operated on and not hypoprothrombinemia induced by the use of acetylsalicylic acid or salicylic acid chewing gum.

Deficiency of vitamin C has, likewise, been demonstrated as a contributing factor in tonsillar hemorrhage and epistaxis. Neivert [6] and his associates found that 27 patients out of a total of 36 suffering from recurrent epistaxis were improved by a daily dose of 300 mg. of ascorbic acid and 15 mg. of vitamin K given by mouth. The combination of vitamin C and vitamin K seems to have a much more beneficial effect than either vitamin alone. In all of the patients treated, the prothrombin time and ascorbic acid content of the blood were below normal.

SUMMARY AND CONCLUSIONS

All bleeding of the nose and throat deserves prompt, careful investigation by someone who appreciates its importance.

When the bleeding source has been located, methods for controlling it effectively should be instituted immediately and continued until all danger has passed. A child who has been having extensive hemorrhage should not be permitted to receive an inhalation anesthetic until he has received an adequate transfusion of whole blood and his hemoglobin level has been accurately determined. Oxidized cellulose dressings are very effective in controlling both nose and throat hemorrhage. They are easy to apply and are absorbed readily.

Vitamin C (ascorbic acid) and vitamin K combined, without the use of acetylsalicylic acid and salicylic acid chewing gum, seems to give the patient convalescing from a tonsillectomy and adenoidectomy the best chance to recover without any complicating bleeding.

1930 Chestnut St.

6. Neivert, H.; Engleberg, R., and Pirk, L. A.: Nasal Hemorrhage: Studies of Ascorbic Acid, Prothrombin and Vitamin K, Arch. Otolaryng. **47**:37-45 (Jan.) 1948.

RHINOPLASTIC APPROACH TO CERTAIN RHINOLOGICAL PROCEDURES

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ALTHOUGH the history of rhinoplasty dates back to antiquity, it is only during the past several years that rhinoplasty has been used as an approach as well as a part of rhinologic procedures.

The rhinoplastic surgical approach is defined as that surgical procedure involving any, many, or all of the components of the nasal pyramid which is performed in such a way that the repair or restoration of any of the involved parts will give the best esthetic and physiological results.

The rhinologic problems that are to be considered involve the external pyramid itself, its inner nasal component—the septum—and, finally, a combination of these two. This article is confined primarily to those problems in which these parts interfere with the free passage of air into the nares and cause obstructions to nasal secretions. In an analysis of these problems, it is necessary to consider the nose in relation to all adjacent structures, such as the upper lip and the eyes.

Rhinoplastic approaches to the external nasal framework may be performed extranasally, through an external incision or incisions, or intranasally, through an internal incision or incisions. Since the intranasal approach may be performed as aseptically as the extranasal and since no visible scar is present, it is naturally the method of choice. The extranasal approach is used when the pathological condition involves the superficial structures of the nasal pyramid, such as direct traumatic wounds, scars, or neoplasms. The approach to the inner nasal components depends upon the intranasal pathology. If the pathology involves the septum alone, either the caudal or the cephalic approach may be used. However, if the pathology involves the external nasal pyramid as well as the septum, then the procedure used would combine the two approaches to the intranasal and extranasal operation and the precise method used would depend primarily upon the pathology of the upper lateral cartilaginous components: the septum and upper lateral cartilages.

The accompanying table illustrates graphically the problems and the procedures that are followed.

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EXTERNAL NASAL PYRAMID

Reconstruction of the external nasal pyramid may be divided into three parts.

A. Covering.—As previously stated, the extranasal approach is used to correct pathological conditions involving the superficial structures or covering. The involvement may be slight or extensive and may occur over the osseous or upper cartilaginous or lower cartilaginous vaults, or any combination of these three areas. Partial involvement is repaired, if possible, by coapting the adjacent structures and then suturing, or by undermining these structures and sliding the tissues so that they can be sutured after they coapt. If too great a deficiency of the nasal covering exists, then either a free, full, or split thickness graft, or a pedicle, or a sliding flap may be used. In complete or almost complete deficiencies of the covering, the pedicle or flap methods may be used. These may be of two general types: those from immediately adjacent areas, such as the Indian [1] and

Problems and Procedures in Rhinoplastic Surgical Approach

Location	Approach
I. External nasal pyramid	
A. Covering	Upper rhinotomy
B. Framework	Upper rhinotomy Septotomy Lower rhinotomy
C. Framework and covering	Upper rhinotomy Septotomy Lower rhinotomy
II. External nasal pyramid and septum	
A. Above the critical line	
1. Osseous vault, cephalic portion of the septum	Cephalic rhinoplasty
2. Upper cartilaginous vault and cephalic portion of the septum	Cephalic rhinoplasty
B. Below the critical line	
1. Upper cartilaginous vault at critical line and caudal end of the septum	Caudal rhinoplasty
2. Lower cartilaginous vault and caudal end of the septum	Caudal rhinoplasty
C. Above and below the critical line	
1. Osseous vault and caudal portion of the septum	Caudal rhinoplasty
2. Upper cartilaginous vault and caudal portion of the septum	Caudal rhinoplasty
3. Lower cartilaginous vault and cephalic portion of the septum	Cephalic rhinoplasty

French [2] flaps; or those from remote areas, such as the Italian [3] and advancing pedicle flaps.[4] If not too great a deficiency exists, split [5] or full thickness grafts may be used. If no pathological condition of the nasal covering is present, then the internal approach is the method of choice. This is performed in two or three stages: upper rhinotomy, septotomy, and lower rhinotomy. The upper rhinotomy, or first stage, involves a separation of the skin from the upper cartilaginous and bony vaults by means of a direct incision through the limen vestibuli.[6] The skin and muscle covering are elevated by means of double-edged knives or scissors. The periosteum over the bony vault may or may not be elevated, depending upon

1. Fomon, S.: Surgery of Injury and Plastic Repair, Baltimore, William Wood & Company, 1940, p. 242.

2. Larrey, cited by Kolle, F. S.: Plastic and Cosmetic Surgery, New York, D. Appleton and Company, 1911.

3. Fomon,[1] p. 243.

4. Roux, P. J.: Quarante années de pratique chirurgicale, Paris, W. Masson, 1854.

5. Blair, V. P., and Brown, J. B.: The Use and Uses of Large Split Skin Grafts of Intermediate Thickness, Surg., Gynec. & Obst. **49**:82, 1929.

6. Blair, V. P., and Brown, J. S.: Nasal Abnormalities, Fancied and Real; Reaction of Patient; Their Attempted Corrections, Surg., Gynec. & Obst. **53**:797, 1931.

the operator's choice. This is followed by the second stage, the septotomy—a procedure in which the skin and the columella are separated from the ventral and caudal surface of the septum by a blunt-end knife which hugs the septal margin ventrally and caudally up to the nasal spine. At this point its detachment from the spine is completed with a Mayo scissors. The third stage, or lower rhinotomy, may be performed now, or it may be deferred until the completion of the surgery of the framework. This involves elevating the skin over the lower lateral cartilages by means of incisions along the inferior border and the elevation of the skin with blunt-edged scissors up to the previous upper rhinotomy incision at the limen vestibuli. Whatever the surgery, an upper or lower rhinotomy and/or septotomy, or a combination of any of these procedures, must be performed.

After these three procedures, which primarily involve exposure of the skeletal or structural components of the external nasal pyramid, are completed, there is the reconstructive surgery of the framework.

B. Framework.—Pathological distortion of the framework may involve the bony, the upper cartilaginous, or the lower cartilaginous vaults, or any combination of these three sectors. Because of the intimate anatomical association of the osseous vault with the upper cartilaginous vault, any pathological distortion in either of these regions may affect the other.

In reconstruction of the osseous vault alone when dealing with a recent fracture, simple manipulation by the use of pressure in the area involved may be sufficient to correct the deformity. However, in old fractures in which the bones must be replaced, an upper rhinotomy followed by a septotomy must be performed before any osteotomies and realignment of fragments. The bony vault is mobilized by means of ventral and lateral osteotomies.[7] The ventral osteotomy separates the nasal bones from one another and from the perpendicular plate of the ethmoid bone and from the nasal spine of the frontal bone. It may be performed by a saw (straight) or a chisel. If a saw is used, the nasal bones are separated from one another and from the perpendicular plate by sawing through the ventral margin of one nasal bone, through the perpendicular plate, and then through the other nasal bone. This may be performed in one procedure, or by removing the saw after it has cut completely or partially through the perpendicular plate and then reinserting a saw at a similar proper level in the other nasal bone and cutting through to join the previously cut area. The blunt-end knife is then used to free the hump from its attachment to the upper cartilaginous vault. (If a chisel is used, each nasal bone is separated from the junction of the perpendicular plate and its nasal crest and the nasal spine of the frontal bone.[8]) After this, the lateral osteotomy is performed.[9] This involves incising the mucous membrane at the pyriform sinus margin. The periosteum is elevated over the nasofacial groove at the ascending process of the maxilla and a right-angle saw or chisel is inserted, depending upon the surgeon's choice. The bones are then separated at this area, care being taken not to perforate the mucous membrane lining of the nose. After

7. Joseph, J.: Nasenplastik und sonstige Gesichtsplastik: Nebst einem Anhang über Mammonplastik und einige weitere Operationen aus dem Gebiete der äusseren Körperplastik, Leipzig, Curt Kabitzsch, 1931 (Pt. 3), p. 99.

8. Aufricht, G.: A Few Hints and Surgical Details in Rhinoplasty, Laryngoscope **53**:317, 1943.

9. Joseph,[7] p. 100.

both nasal bones are separated from their attachments at the nasofacial groove, direct pressure on the nasal bone from without may be used to loosen the fragments from the nasal notch, or chisel outfracture from the ventral margin may be performed.

The upper lateral vault is approached through an upper rhinotomy followed by a septotomy. After exposure of the upper laterals, the following procedures may be performed: (*a*) intranasal chondrotomy—direct incision of the mucous membrane through the upper lateral cartilages at their junction with the septum (the skin being protected from this incision by the use of an angular retractor); (*b*) submucous chondrotomy—incision of the upper lateral cartilages at their junction with the septum after the mucous membrane and perichondrium have been detached submucously (mucous membrane being left intact); or (*c*) simple chondrotomy—the cartilage is cut from the ventral surface toward, but not including, the mucous membrane.

After exposing the upper lateral cartilages through the indicated procedure, the trimming is performed. The type of upper lateral surgical procedure to be used will depend upon whether or not any septal surgery is performed, and, if so, upon its extent and location.

Because of the intimate anatomic relationship between the osseous and upper lateral vaults, there are many problems common to these two areas. These may be classified as hump or saddle or shifting deformities or any combination of the three. Hump, shifting, or a combination of hump and shifting deformities are approached through the upper rhinotomy-septotomy route. After removal of the hump, the indicated chondrotomy may be performed. This is followed by the lateral osteotomies. Trimming of the upper laterals may be performed now or deferred until after treatment of the lower cartilaginous vault. Shifting deformities of both upper vaults may be mild or marked. The mild deformities are treated like hump deformities. In the case of marked shifting of the nose, a wedge of bone may be removed from the wider side so as to compensate for this deformity. Saddles may be approached in one of the two ways, depending upon the presence or absence of pathology in the lower lateral vault. If there is need for lower lateral reconstruction, then an upper rhinotomy is followed by a complete septotomy. After a lower rhinotomy with the subsequent reconstruction of the tip, the graft [10] is inserted through the lower rhinotomy incision. If there is no need for lower lateral reconstruction, then the upper and lower rhinotomies are performed with a partial septotomy (only over the dorsum), and, finally, with insertion of the graft over the saddle area. Simulated saddles are treated like hump deformities.

Since the lower vault and its adjoining upper lip form the boundaries of the nares, they must be considered as one anatomic unit. The nares, or nostrils, are bounded laterally by the ala, anteriorly or ventrally by the angle, medially by the columella, posteriorly by the floor or nasolabial junction. Nostril deformities are seldom due to a single defect in its boundaries, but rather to the sum of all of them. In some cases, the nostril deformity may be due to causes outside its boundaries, such as an elongated or foreshortened septum or upper lateral carti-

10. Pierce, G. W., and O'Connor, G. B.: Reconstruction Surgery of the Nose, Ann. Otol. Rhin. & Laryng. **47**:437, 1938.

lages, or both. Nostrils may be wide or narrow, long or short, tilted up or down, or twisted, or may show any combination of these characteristics. Deformities of the nostrils due to lower lateral cartilages and deformities of the nasal tip are approached through the lower rhinotomy. This procedure generally follows the previously described upper rhinotomy and septotomy. After the lower lateral cartilages are freed with a blunt-end scissors they are trimmed. Whatever the procedure of trimming, the angle or junction of the medial and lateral crura is generally incised either submucously or directly and then the cartilage is trimmed. Care should be exercised in conserving the vestibular lining. The ala cartilages and angle are corrected by removing strips of the upper border and the cut angle margin, depending upon the extent of deformity. After this is done, the vestibular lining is trimmed to size, if too large a vestibule is present. Frequently, the cut end of the angle margin is not trimmed, to permit it to lie over the medial crura. In this case, its vestibular skin is excised. The medial crura then are delivered through the lower rhinotomy incision and corrected as indicated—for example, by trimming the angle margin or removing the intercartilaginous connective tissue in order to narrow the columella. The base of the columella is thinned out by removing any excess connective tissue and suturing the columella base. A hanging columella may be corrected through this approach by removing a projecting caudal end of the septum or an excessive membranous septum or protruding nasal spine. The retracted columella may be corrected by using struts. However, the floor or nasolabial junction, as well as the columella length and the base of the columella, is approached through the lower part of the septotomy incision and through a premaxilla incision. The premaxilla incision is an extension of the lower portion of the septotomy in the direction of the ala-facial junction. If greater mobility of the upper lip is desired, the ala may be detached at the ala-facial junction and the premaxillary incision is extended up to it. This permits greater mobility of the upper lip and will allow for either the building up of a short columella at the expense of a large upper lip or the recession of an elongated columella into a short upper lip. This premaxillary incision thus aids in the correction of the harelip nose and the harelip. It converts the upper lip into a sliding graft, thus allowing proper coaptation of the skin and mucous membrane of lip and mouth. It is through this premaxilla incision that a cartilaginous implant or a graft may be inserted to build up a recessed premaxillary area.

Deformities involving all three vaults are not uncommon. Procedures involving correction of both upper vaults invariably require correction of the lowest segment, and vice versa. The procedure of correcting all the vaults of the nasal pyramid is known as a total rhinoplasty. This consists of an upper rhinotomy, septotomy, ventral osteotomy or a hump removal, intranasal, submucous, or simple chondrotomy, and trimming of the upper lateral cartilages, bilateral osteotomies, and, finally, a lower rhinotomy and trimming of the lower laterals. This procedure is followed in hump, shifting, or hump and shifting deformities. However, saddle deformities generally are corrected through the upper rhinotomy, septotomy, lower rhinotomy, trimming of the lower lateral cartilages, and, finally, graft implant.

C. Framework and Covering.—Deformities involving both the framework and the covering are treated by a combination of procedures involving both these structures.

EXTERNAL NASAL PYRAMID AND SEPTUM

The intimate anatomicopathological relationship that exists between the framework and its contiguous intranasal structure, the septum, makes this area one of the most difficult plastic and rhinologic problems to treat. It has been the heartache of the rhinologist, who, having performed a successful septal operation, finds that either his patient is still complaining of obstructed breathing due to a deformed external pyramid or else that in his patient a saddle nose has developed at the weak spot. Likewise, even though the surgeon has produced a commendable cosmetic result on the external pyramid, he has, by relocating the lower nasal vault, added an intranasal obstruction to a previously questionable unobstructed passageway—a previously adequate septum has become inadequate. The approach to the nasal pyramid and the septum must be performed in such a manner that the support and projection of the external pyramid are properly maintained and the septum maintains its proper configuration and location for optimum physiological function. The support and projection of the nasal dorsum is maintained primarily by the osseous and upper cartilaginous vault. The lower cartilaginous vault hangs as an island of cartilage in the nasal lobule and it adds firmness to it (just as starch does to a collar or a cuff). The projection and supporting ability of the nasal lobule is directly proportional to the firmness and size of its lower cartilaginous vault structure, the caudal end of the septum, and the nasal spine. The osseous vault is firmly supported by the nature of its structural components. However, the upper cartilaginous vault is dependent primarily upon the support given to it by its adjacent structures. If the upper lateral cartilages are detached from any of these structures, some weakness develops. Since the upper lateral cartilages are angulated projections of the quadrilateral cartilage, their junction and support are firm. The cephalic portion of the upper cartilaginous vault is attached to the under surface of the osseous vault, and it acts as its cantilever arm [11]; thus it is more firmly attached than its free caudal portion, which projects under the lower cartilaginous vault and assists in its support. The firmness of projection of the caudal portion of the upper lateral cartilage depends on the over-all length of the upper lateral cartilages as compared with the length of its septal attachment. It also is dependent upon the acuteness of the angle at which the upper lateral cartilages swing down and away from the caudal end of their septal attachment. It is at this point that the fascial attachment of the upper lateral cartilages to the lower lateral cartilages is found. This is the most poorly supported area of the dorsum and is the area of the dorsal "weak spot." [12] Since it is essential that the dorsum be maintained, prime consideration must be given to the support of its weakest portion—the weak spot and the immediately adjacent upper cartilaginous vault. Since this weak spot is the caudal-ventral end of the septal attachment and the nasal spine is the caudal-dorsal end of the septal attachment, any portion of the septum projecting ventral and caudal to an imaginary line connecting these two points is considered the suspended or caudal portion of the septum. This imaginary line, which is called the critical line, separates the

11. Fomon, S.; Syracuse, V. R.; Boloton, N., and Pullen, M.: Plastic Repair of the Deflected Nasal Septum, Arch. Otolaryng. **44**:141 (Aug.) 1946.

12. Fomon, S.; Gilbert, J.; Silver, A., and Syracuse, V.: Plastic Repair of the Obstructing Nasal Septum, Arch. Otolaryng. **47**:7 (Jan.) 1948.

cephalic portion from the caudal portion of the septum. The caudal portion is made up of nasal spine and quadrilateral cartilage. The cephalic portion is composed of the remaining portion of the quadrilateral cartilage, the perpendicular plate, the vomer, and all contiguous bony junctions. However, the cephalic portion which is to be considered has a direct effect on the nasal pyramid and lies directly in front of the anterior border of the interior turbinate. Thus the structural function of the caudal end of the septum is to aid in projecting the lower cartilaginous vault and the columella. Similarly, the function of the cephalic portion of the septum is to aid in projecting the upper cartilaginous vault up to its adjacent weak spot. Thus, when any combined surgery (external pyramid and septal) is done, the rhinoplastic approaches will have to depend on whether the septal deformity lies ventral and caudal, or dorsal and cephalic, to the imaginary critical line. If the septal surgery is to be above the critical line, the cephalic approach is used; if below the critical line, the caudal approach is used. The cephalic approach involves a septal surgical approach above the critical line plus a rhinoplasty in which an intranasal chondrotomy is performed. The caudal approach involves a septal surgical approach below the critical line plus a rhinoplasty in which either a simple or a submucous chondrotomy is performed.

Before any of the reconstructive problems and their treatment are considered, it would be wise to classify the various types of septal operations. A simple classification can be based on the amount of septal structural covering (mucous membrane, perichondrium, and periosteum), location of incision (whether right or left side and whether above or below the critical line), and, finally, the amount and location of the septal supporting structures removed. Thus, if the entire septal covering from the caudal end is elevated up to and beyond the line of the anterior border of the inferior turbinate, this is known as unilateral total septotomy. If this has occurred on the right or left or both sides, it would be known as a right or left or bilateral total septotomy. If part of the mucous membrane is elevated on one side, this is a partial septotomy; similarly, right or left or bilateral. In these cases if the area of the incision is located above the critical line, it would be known as cephalic; if below or at the critical line, it is caudal. If the incision is at the caudal end of the septum, this would be a total septotomy. If the entire septum is removed, this is a total septectomy. If a portion is removed, it is a partial septectomy. Thus the Fomon operation [4] can be described as a bilateral total septotomy with a total or partial septectomy with a caudal or caudal-cephalic cartilaginous implant. The Galloway operation [4] is a right or left total septotomy and partial septotomy, right or left, with a partial septectomy. The Killian operation is a bilateral partial right or left cephalic septotomy with a partial septectomy. (Right cephalic would indicate the incision was made in the mucous membrane above the critical line; right caudal would indicate the incision was made at or below the critical line.) The Metzenbaum operation [13] is a unilateral total septotomy, right or left, with cross-hatching of the caudal cartilaginous area.

13. Metzenbaum, M. F.: Recent Fracture of Nasal Base Lines of Both Outer Nasal Walls with Divergent Displacement: Orthopedic Procedures for Obtaining Anatomic Reduction of Osseous and Cartilaginous Nasal Framework, Arch. Otolaryng. **34**:723 (Oct.) 1941.

The problems of reconstruction of the framework and the septum thus may be divided into those located above the critical line, those below it, and a combination of the two.

A. Problems Arising Above Critical Line.—These may be divided into three groups: (1) problems involving the osseous vault, nasal spine of the frontal bone, and the cephalic portion of the septum; (2) problems arising in the upper cartilaginous vault and the cephalic portion of the septum; (3) problems arising in the osseous and cartilaginous vault and cephalic portion of the septum.

B. Problems Arising Below Critical Line.—These may be divided into two groups: (1) problems involving the upper cartilaginous vault, at the critical line, and the caudal end of the septum; (2) problems involving the lower cartilaginous vault and the caudal end of the septum.

C. Problems Arising Above and Below Critical Line.—The same close anatomic relationship that exists between the osseous vault and the upper cartilaginous vault extends to their appendages in the septum. A deformity of one may extend to a part or the whole of the other, and vice versa. Thus, an injury of the osseous vault and the perpendicular plate of the ethmoid bone may extend to the upper cartilaginous vault, with or without its quadrilateral cartilage. Similarly, an injury to the upper cartilaginous vault alone may extend to the osseous vault with or without the perpendicular plate, or may extend from the upper cartilaginous vault to the perpendicular plate of the ethmoid bone, and so on. The following areas may be involved: (1) the osseous vault and caudal portion of the septum; (2) the upper cartilaginous vault and caudal portion of the septum; (3) the lower cartilaginous vault and cephalic portion of the septum; (4) the lower and upper cartilaginous vault and osseous vault with involvement of caudal and cephalic portions of the septum.

Problems arising above the critical line and involving the osseous vault and cephalic portion of the septum are treated through the cephalic rhinoplastic approach. The external pyramid surgery is the same as the surgery of the osseous vault in which an intranasal chondrotomy is performed. The correction of the septum involves a bilateral partial right or left cephalic septotomy followed by a partial septectomy; the cephalic portion of the septum may be removed entirely, if necessary. It makes no difference whether the septal surgery or osseous vault surgery is performed first. This may be performed either to correct a severe recent fracture or to reconstruct some old pathology. It may also be performed as part of a rhinoplasty in which a cosmetic septal operation has to be performed. In a cosmetic septum, the ventral portion of the perpendicular plate and nasal spine of the frontal bone are removed in order to permit sufficient narrowing of the external nasal pyramid. Since in this operation the septal surgery performed lies above the critical line, the approach is cephalic.

Problems arising above the critical line and involving the upper cartilaginous vault and the cephalic portion of the septum are treated by the cephalic rhinoplastic approach. Since the upper lateral cartilages and the quadrilateral cartilage of the septum are anatomically one structure, any injury to one part almost invariably affects the rest. Because of this, the reconstruction of this region presents combined and individual problems. The upper lateral cartilages are considered as to their length, width, projection, location, and configuration, and, finally, as to their

being weakly or strongly supported. The septum must be considered as to its length, thickness, location, configuration, and, finally, as to its firmness. Problems arising from the combination of the upper lateral cartilages and the quadrilateral cartilage involve the volume of their intranasal airway and the supporting strength of the middle vault dorsum. The intranasal volume at the upper cartilaginous vault depends on the acuteness of the angles (from the intranasal aspect) at which both upper lateral cartilages meet the septum and on the thickness, location, and configuration of the septal cartilage. The length, width, location, projection, and configuration of the upper lateral cartilages can all be corrected by the rhinoplastic procedures described under upper cartilaginous vault reconstruction, the chondrotomy (intranasal or submucous or simple) being followed by trimming. The projecting strength of the upper lateral cartilage depends upon all of its adjoining supporting structures, particularly its septal component, the quadrilateral cartilage. If a septectomy or septal correction is performed at the critical line or ventral or caudal to it, an intranasal chondrotomy is almost always ruled out. An intranasal chondrotomy removes whatever support the septum gives to the upper cartilaginous vault. Thus, in these procedures the simple or submucous chondrotomy is the method of choice, because it, at least, leaves an intact mucous membrane between the septum and the upper lateral cartilage and releases the spring action at this juncture. If an intranasal chondrotomy has been performed and the dorsum collapses, the upper lateral cartilages may be sutured to one another. If, after they are sutured together, the dorsum is still somewhat poorly projected, a strip of cartilage or bone may be implanted above it. Thus, as a rule, in procedures which are cephalic or dorsal or both cephalic and dorsal to the critical line, an intranasal chondrotomy is the method of choice. In procedures caudal or ventral, or caudal and ventral to the critical line, the simple or submucous chondrotomy is the method of choice. The length, thickness, location, configuration, and firmness of the septum are all corrected through the lateral septotomy and septectomy. It must not be forgotten that the cephalic portion of the septum aids in supporting and projecting the upper cartilaginous and osseous dorsum, while the caudal portion of the septum assists in the projection of the columella and the membranous septum. In combined surgery the upper cartilaginous vault dorsum will maintain its projection when the cephalic portion of the septum is removed, because the septal support along the critical line remains intact to maintain the weak spot. In general, in this group where the problems involve reconstruction of the upper cartilaginous vault and the cephalic portion of the septum, the cephalic approach is used. This entails surgery of the upper cartilaginous vault with an intranasal chondrotomy, and a cephalic approach to the septum is to be used.

Problems arising below the critical line and involving the lower or upper cartilaginous vault, or both, and the caudal end of the septum are treated by the caudal rhinoplastic approach. The lower cartilaginous vault surgery is performed as previously described under framework reconstruction. When the upper lateral cartilages are involved at the level of the critical line or the weak spot, a simple or submucous chondrotomy is performed after exposure of the upper lateral cartilages. The septum is exposed through the caudal or total septotomy incision. The caudal portion of the septum must be treated in such a manner as to main-

tain the projection of the columella and of the weak spot. If it is excised entirely because of its twisted nature, other cartilage must replace it either as a single larger plaque or as two smaller struts, so as to avoid a shortening or retraction of the columella and a collapse of the weak spot. If only a small strip of the caudal end of the septum has to be removed and the columella alone needs to be projected, then a strut of cartilage in the columella may be sufficient. If the weak spot has collapsed, then a strut or plaque of cartilage must be inserted to maintain its support. It is not advisable to use a single cartilage both to project the columella and to support the weak spot, because this piece of cartilage will eliminate the membranous septum.

Problems arising both above and below the critical line are treated by either the cephalic rhinoplastic approach or the caudal rhinoplastic approach. The method of choice depends upon the type of septal surgery. If the septal surgery is cephalic, then the former is employed; if caudal, the latter is employed.

SUMMARY AND CONCLUSIONS

The approach to the external pyramid is best performed through the intranasal route—the upper rhinotomy, septotomy, and lower rhinotomy.

A simplification of septal surgery classification has been offered. The surgery of the upper lateral complex—upper lateral cartilages and septum—depends upon the location of the septal defect. If cephalic septal surgery is performed, an intranasal chondrotomy is indicated. If caudal septal surgery is performed, a simple or submucous chondrotomy is performed. The approach to external pyramid and septal surgery is either through the cephalic rhinoplastic approach or through the caudal rhinoplastic approach.

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MECHANISM OF SHADOW HEARING

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THE AUDIOGRAM recorded in instances of monaural deafness when the receiver is placed at the deaf ear is called the "shadow curve." Apparent hearing with the deaf ear is called "shadow hearing."

It seems that the phenomenon of shadow hearing was known to pioneers of otology and in the era preceding the invention of the audiometer. Bezold [1] made the remark, "Hearing which appears to be in the deaf ear represents in reality an attenuated reflection (*ein abgeschwächtes Spiegelbild*) of the hearing in the healthy ear." The factor instrumental in the process of shadow hearing is generally regarded as the transmission of sound waves to the opposite side across bone. According to Bunch,[2] however, the sound can be heard through or around the head, i. e., by bone or by air conduction. This concept appears refuted by the fact that the tone is greatly enhanced when a finger is placed in the good ear, an observation mentioned by Fletcher.[3]

Saltzman in his "Clinical Audiology" [4] maintains that at an intensity of 50 db.[4a] or 30 db.[4b] the vibrations are sufficiently powerful to traverse the 21 cm. of skull, the distance from one ear to the other. His statement appears to be supported in a recent article by Saltzman and Ersner.[5]

PRESENT STUDIES

In the present report information on the factors responsible for the mechanism of shadow hearing is presented. This material is the result of studies on the subject in both the monaurally deaf and the normal person. By means of an experimental arrangement, conditions were established in the normal subject which were regarded as approximately analogous with those responsible for shadow hearing in the monaurally deaf.

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1. Denker, A., and Kahler, O., editors: Handbuch der Hals- Nasen- Ohren- Heilkunde, Berlin, Julius Springer, 1926, Vol. 8, p. 588.

2. Bunch, C. C.: Clinical Audiometry, St. Louis, C. V. Mosby Company, 1943, pp. 54-55.

3. Fletcher, H.: Speech and Hearing, New York, D. Van Nostrand Company, Inc., 1929, p. 174.

4. Saltzman, M.: Clinical Audiology, New York, Grune & Stratton, Inc., 1949, (*a*), p. 6; (*b*), p. 155.

5. Saltzman, M., and Ersner, M. S.: Masking and Shadow Hearing in Bone Conduction, Arch. Otolaryng. **51**:809-813 (June) 1950.

PROCESS OF SHADOW HEARING

The process of shadow hearing when the air receiver is attached to the deaf ear may be conceived to consist of three phases:

In the first phase sound waves introduced by air conduction into the external meatus of the deaf ear are transmitted to the adjacent bony structures of the same side, thus initiating the so-called "indirect bone conduction."

In the second phase these sound waves travel through the skull to the opposite petrous bone.

In the third phase these sound waves enter from the petrous bone of the good side into the inner ear. This phase, of course, is not specific for the process of shadow hearing, since it is equally present when sound waves in the petrous bone originate from an ipsilateral or a contralateral bone-conducted sound source. It goes without saying that the shadow curve recorded with the air receiver attached to the deaf ear depends on factors involved in both the first and the second phase, while the shadow curve recorded with the bone receiver at the deaf side depends on factors involved in the second phase only.

THE PROBLEM

The problem at issue revolves around two questions: 1. Which of the first two phases is responsible for the tonal loss significant for shadow hearing? 2. If both contribute to this effect, how great, in numbers of decibels, is the share of each individual phase in this process?

STUDIES ON THE MONAURALLY DEAF

The opportunity presented itself to study two patients with complete monaural deafness and approximately normal hearing in the good ear. The audiograms recorded on these patients conformed with each other except for a few minor inconsistent and insignificant differences. A representative case is that of a man aged 61, whose audiogram is shown in Chart 1. His deafness resulted from an acoustic neuroma on the left, operated on 12 years previously. Both ear drums were normal. There was paralysis of the left facial nerve, partly repaired by a plastic procedure performed nine years previously. No caloric response was obtained at the left. His audiograms were taken with the Maico audiometer (H. I.) in a quiet but not soundproof room.

OBSERVATIONS

Six curves are presented in the audiogram (Chart 1). They represent, in sequence from top to bottom, the following experimental conditions: (1) air receiver at the right ear; (2) bone receiver at the left mastoid with the right ear occluded by the finger or by a telephone receiver (an almost identical curve was recorded when the bone receiver was placed at the right mastoid with equivalent conditions maintained); (3) bone receiver at the right mastoid with the right ear open; (4) bone receiver at the left mastoid with the right ear open; (5) air receiver at the left ear with the right ear occluded; (6) air receiver at the left ear with the right ear open.

Comment

The air-receiver audiogram recorded from the left ear with the right ear open (Curve 6) shows an elevation of the threshold over the one recorded from the right (normal) ear (Curve 1) of 55 to 75 db. The elevation of the threshold in the left ear when the right ear was closed (Curve 5) is 35 to 60 db. These findings conform to the observation of Fletcher, mentioned above. They indicate that in monaural deafness hearing in the deaf ear depends on stimulation of the good ear by sound waves which travel across the bone rather than around the head through the air, a medium which Bunch considered as an additional possibility. If the waves were transmitted by the latter route, closing the good ear should cause an elevation, rather than a drop, of the threshold for shadow hearing. In this connection, attention may also be called to the pattern of the two shadow curves in correlation with each other. The depression of the threshold in the lower frequencies with a gradual elevation in the higher ones, as seen in Curve 5, is charac-

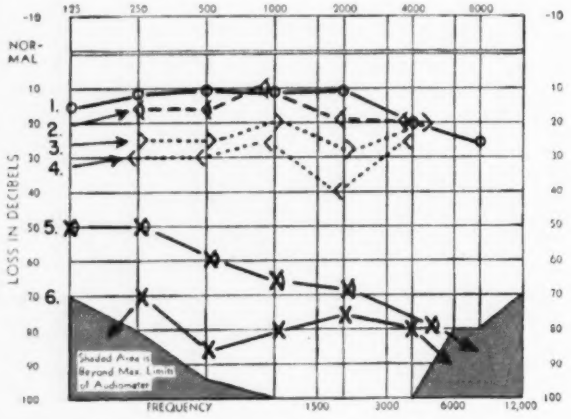

Chart 1.—Representative audiogram of a man of 61 with deafness in the left ear. For explanation see text.

teristic of the Weber phenomenon, which, as we know, manifests itself more in the lower than in the higher pitches. The audiogram recorded with the bone receiver at the deaf ear (Curve 4) shows an elevation of the threshold over the one recorded with the bone receiver at the good ear (Curve 3) of approximately only 5 to 10 db. Moreover, even this small difference was not recorded when, in either one of these two positions, the good ear was kept closed (Curve 2).

The threshold differences recorded with the bone receiver at the right (without masking—Curve 3) and those recorded with the bone receiver at the left (without masking — Curve 4) do not agree with the statement of Saltzman that a sound pressure of 30 or of 50 db. is sufficient to traverse the 21 cm. from one ear to the other to stimulate the healthy organ. In the reported case, as in another case, an increase of the sound pressure by only 5 to 10 db. appeared sufficient to give this effect.

On the basis of these figures the tonal loss which is to be attributed to the first phase (i. e., transition of sound waves from air into bone), when calculated from the difference between the total tonal loss and the loss depending on the second phase (i. e., travel of sound waves across the bone), equals 55 to 75 db. minus 5 to 10 db.

Answers to the Problem

The two questions raised earlier in this article may be answered thus:

1. Evidence is presented that both the first and the second phase contribute to the total tonal loss in the process of shadow hearing. In other words, both the transition of air-conducted sounds into the bone and the travel of bone-conducted sound waves across the skull have their share in the phenomenon.

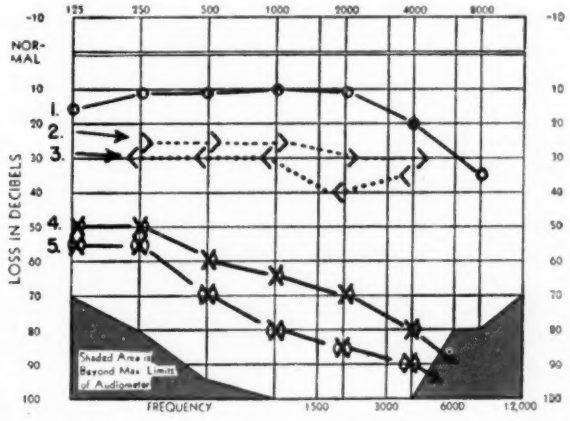

Chart 2.—Audiogram of the same subject. Notice the difference between the shadow curves recorded with the air receiver when the good ear is closed and the deaf ear open (Curve 4) and when the good ear is closed and the deaf ear plugged (Curve 5). The shadow curve recorded with the bone receiver at the deaf mastoid is the same (Curve 3) whether the deaf ear is plugged or kept open.

2. The first phase contributes to the total tonal loss about 50 (45) to 70 (65) db. The share of the second phase is 5 to 10 db. These figures indicate that the tonal loss is mainly caused by transition of sound waves from air into bone.

Conditions in the Deaf Ear of Possible Influence on Shadow Hearing

Shadow hearing is to be regarded as a manifestation of the auditory function of the good ear. It can be shown, however, that factors involving conditions in the deaf ear may also be of influence in the process. This influence is evidenced in the following experiment:

The external meatus of the deaf ear was plugged with impression material, such as that ordinarily used for ear molds. The good ear was tightly closed by an earphone in order to prevent stimulation by air-conducted sound waves possibly traveling around the head when strongest intensities were applied. Chart 2 shows that the shadow curve recorded with the deaf ear plugged and the right ear closed (Curve 5) is considerably elevated above the curve recorded with the deaf ear

unplugged (Curve 4). No essential changes in the respective shadow curves produced by plugging the deaf ear were noted when the bone receiver was applied (Curves 2 and 3).

Law Governing Threshold of the Shadow Curve

Correlation of this experiment with that of Fletcher presents evidence that in shadow hearing the threshold of the shadow curve appears to be influenced as follows: Impedance in the deaf ear elevates the threshold of the shadow curve; impedance in the good ear lowers it.

Experiments on Persons with Normal Hearing

An attempt was made to check the results of the foregoing studies by experiments performed on subjects with normal hearing under conditions presenting an approximate analogy to those fundamentally responsible for the process of shadow hearing in the monaurally deaf. It can be conceived that the main factor responsible in this process is the impedance caused by the mass of the subject's head being intermediate between the sound source located at the side of the deaf ear and the stimulated end-organ at the opposite side.

It was assumed that an analogy to this situation could be established by placing a human head between the ear of a normal observer and the sound source. Such an arrangement creates experimentally in the normal person an impedance factor which can be regarded as approximately equal to the one represented in the monaurally deaf subject by his own head. Some technical difficulties faced in the beginning of our experimentations were circumvented by the use of an ordinary stethoscope.

The idea of using the stethoscope for audiological studies is not new. It was originated by authorities such as Harless, Mader, Bezold, Claus, and others [6] in their investigations on skull resonance half a century ago. A review of the literature did not reveal a report referring to the application of the stethoscope in studies concerning the process of shadow hearing.

Acoustic Conditions Peculiar to the Stethoscope

It seemed advisable to have these experiments preceded by studies on the differences between acoustic conditions associated with the use of the binaural stethoscope and those associated with monaural hearing when the air receiver is directly attached to the external ear. Furthermore, the use of the open bell and the diaphragm in this procedure, and the binaural versus the monaural application of the stethoscope, were correlated. The assistance of four experienced observers was helpful in these studies.

Chart 3 shows a representative audiogram recorded on a man of 27 with bilaterally equal normal hearing, as indicated by the heavy line. With the diaphragm attached to the air receiver, the threshold of hearing, as correlated with that recorded by direct application of the air receiver to the ear, appears to be raised throughout the entire spectrum, particularly in the higher frequencies. With the bell attached to the air receiver again, the threshold in the higher frequencies is raised to a smaller degree, however, while in the lower ones a slight drop of the threshold is noticed.

6. Denker and Kahler,[3] Vol. 6, pp. 433-442.

There was only an inconsistent and rather fluctuant difference, which was noted particularly in the upper frequencies when one branch of the tubing was clamped by a hemostat or separated from the external meatus. These findings explain the preference of physicians for the diaphragm over the bell when attempting to pick up high-pitched murmurs. It appears that with attenuation of the lower notes with the diaphragm, the masking effect of lower pitches on the higher ones is diminished, as pointed out by Rappaport and Sprague.[7] On the basis of these studies I decided to use the open bell in my experiments.

Experimental Procedure

As a routine procedure in these experiments, the following audiograms were recorded: (1) the observer's audiogram for air conduction on each side; (2) his audiogram with the binaural stethoscope placed in his ears and the bell placed at the air receiver (this was termed the "direct stethogram"); (3) his audiogram with

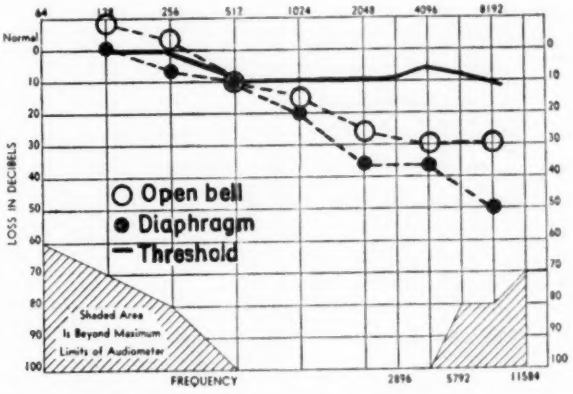

Chart 3.—Representative audiogram of a man of 27 with normal bilaterally equal hearing. "Direct stethograms" are secured with the open bell and the diaphragm respectively.

the air receiver at a subject's external ear and the bell at the ipsilateral mastoid process ("uncrossed cephalostethogram"); (4) the same as in 3; this time, however, with the bell at the subject's contralateral mastoid ("crossed cephalostethogram"); (5-6) the arrangement described in 3 and 4, except that the bone receiver was used instead of the air receiver; (7) crossed air-receiver "cephalostethogram," as described in 4, with the external meatus on the side of the bell closed by a finger or by an air receiver; (8) uncrossed and (9) crossed bone-receiver "cephalostethogram," as described in 5 and 6, with the external meatus closed on the side of the bell.

Observations

The records secured from a representative observer with the procedures as described are presented in Chart 4. Six curves are shown in this audiogram: (1) the observer's routine audiogram, indicated by one line only, since it was approxi-

7. Rappaport, M. B., and Sprague, H. B.: Physiologic and Physical Laws that Govern Auscultation, and Their Clinical Application, Am. Heart J. **21**:257-319 (March) 1941.

mately equal for the two ears; (2) his direct stethogram; (3) his uncrossed bone-receiver "cephalostethogram" recorded with bone receiver and bell both at the subject's left mastoid area; (4) his crossed bone-receiver "cephalostethogram" recorded with the bone receiver at the subject's left mastoid and the bell at his right mastoid; (5) his uncrossed air-receiver "cephalostethogram" recorded with the air receiver at the subject's left ear and the bell at his left mastoid; (6) his crossed air-receiver "cephalostethogram" recorded with the air receiver at the subject's left ear and the bell at the right mastoid.

Findings in positions 7, 8, and 9 are not shown in the audiogram (Chart 4), since they were practically identical with those recorded when the external meatus in the respective positions was kept open.

Comment

The stethograms recorded on the normal person as described show a striking parallelism with the shadow curves recorded on the monaurally deaf. In both a

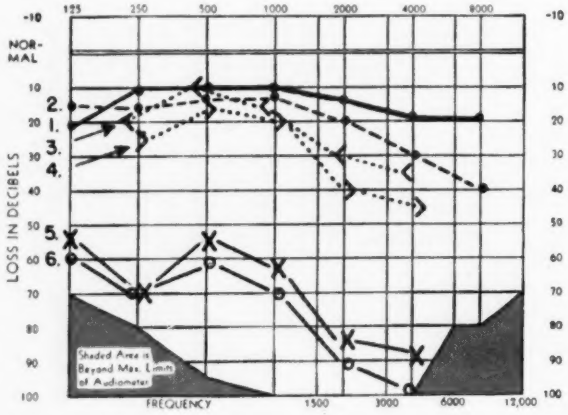

Chart 4.—Audiogram (Curve 1) of a representative subject with "direct stethogram" (Curve 2), uncrossed and crossed bone receiver "cephalostethogram" (Curves 3 and 4, respectively), and uncrossed and crossed air receiver "cephalostethogram" (Curves 5 and 6, respectively).

tonal loss of 5 to 10 db. becomes manifest when the bone receiver is attached to the mastoid opposite the one on which the sound waves are picked up. In the experiment, this was done by the bell (Chart 4, Curve 4); in the monaurally deaf person, by his good ear (Chart 1, Curve 4).

Moreover, the experiment provides the possibility of correlating the records secured by means of the air receiver when the bell is attached at the same mastoid (Curve 5) with the one when it is attached at the opposite mastoid (Curve 6). A tonal loss of 40 to 65 db. appears when the bell is attached to the same mastoid. It is increased by about 5 to 10 db. when the bell is attached to the opposite mastoid. This observation seems of particular interest, since it demonstrates directly that transition of sound waves from the medium of air into one of bone is the main factor responsible for the tonal loss in this process.

In addition, it is shown that the impedance factor represented by the mass of the head (the subject's in the experiment; the patient's in the case of monaural

deafness) is responsible for a tonal loss of only 5 to 10 db. This statement is based on correlation of the crossed with the uncrossed "cephalostethograms" when recorded by application of either the air receiver or the bone receiver. These figures indicate a striking similarity to those resulting from the studies on the monaurally deaf.

REFLECTIONS ON THEORIES CONCERNING WEBER PHENOMENON

Attention may be called to the absence of any significant differences apparent between the "cephalostethograms" secured with the subject's external meatus closed and those recorded with the open ear. In my opinion, this observation indicates that the Weber effect is not dependent on factors responsible for an absolute increase of sound intensity, such as prevention of sound escape or creation of a resonance chamber. If it were so, one would expect the Weber phenomenon to manifest itself by increased loudness, noted equally by the subject and by the observer. On the basis of these experiments I am inclined to believe that factors are responsible for the Weber effect which affect conditions in the subject only and are of no influence on conditions in the observer. Such factors include principally the attenuation of the masking effect caused by ambient noises, and probably changes in the endaural air and/or lymph pressure.

CRITICISM OF EXPERIMENTS

I cannot close this report without calling attention to inaccuracies unavoidably involved in this method of experimentation. First, with the facilities available to me, it was not possible to attach the air receiver and the bell to the underlying structures with equal pressure in all experiments. This unfortunate condition might explain some of the inconsistencies noted in the course of the experiments. Second, owing to leakage in the air receiver, it was not possible to prevent sound waves from entering directly by air into the stethoscope, bypassing the bone. For this reason the possibility had to be considered that hearing recorded in the uncrossed, and probably also in the crossed, "cephalostethograms" depends not on sound waves entering from the air into the bone and therefrom into the stethoscope, but on those slipping through the air receiver and entering the stethoscope outside the bone. I checked on this possibility by correlating audiograms recorded when the bell was attached to the bone with those recorded when the bell was separated from the bone. I anticipated that the latter position would provide better hearing than the former if leakage of sound waves should be the responsible factor and that it would be the reverse in the case that hearing depended on sound waves transmitted from air into the bone. These studies showed in the audiograms recorded with the bell separated from the bone a rise of 10 to 15 db. (more in the lower frequencies) over those with the bell attached to the bone. When the audiograms were recorded in the same manner at the opposite ear, almost nothing was heard with the bell separated from the bone except at one or two of the higher frequencies when intensities of 90 db. or more were applied. Thus, it became evident that the tonal loss appearing in the crossed and uncrossed "cephalostethograms" is due to transition of sound waves from air into bone. It cannot be attributed to sound waves entering the stethoscope from air outside the head as a result of leakage, since this factor, when bone conduction was excluded, appeared insufficient to create an auditory sensation at intensities strong enough for this effect when the sounds were picked up from the bone.

Finally, it cannot be overlooked that in the monaurally deaf, sound waves which arrived at the petrous bone of the healthy side pass directly into the end-organ, while in the experiment they are transmitted from the bone into the stethoscope and then by air into the observer's ears. This factor may detract from the analogy of conditions in these experiments with the conditions prevailing in the monaurally deaf. It cannot, however, be regarded as a distorting variant, since it was constantly and equally present in all parts of the experimental procedure. The results of the experiment show an interesting parallelism with the observations recorded in the monaurally deaf. They are offered not as proof, but merely as support, for the conclusions resulting from the studies on the latter.

SUMMARY AND CONCLUSIONS

An analysis of the process of shadow hearing is presented, based on studies on the monaurally deaf, as well as on the normal subject. In the latter, experimental conditions were established approximating those which are regarded as responsible for the phenomenon in the monaurally deaf. It is shown that the total tonal loss of about 50 to 70 db. significant for the shadow curve appears about equally in the audiogram recorded on the monaurally deaf and that recorded on the normal person under experimental conditions. This tonal loss depends on two factors: (1) the transmission of air-conducted sound waves into the bone; (2) the traveling of bone-conducted sound waves across the skull to the opposite side. Evidence is presented that the second factor contributes only 5 to 10 db. to this effect, which depends mainly on the transition of sound waves from air into bone. This conclusion results indirectly from the audiograms recorded on the monaurally deaf and directly from those recorded in the experimental procedure. Additional studies on the monaurally deaf indicate that the shadow curve appears to be influenced by impedance factors experimentally established in the deaf ear, as well as in the good ear. The effect of impedance appears to obey the law that impedance in the deaf ear raises the threshold of the shadow curve and that impedance in the good ear lowers it.

On the basis of appropriate experimental procedures the Weber phenomenon appears to depend on factors augmenting the loudness sensation in the subject only, but not on factors such as would create an absolute increase of the sound intensity.

Finally, certain variable factors bearing on the accuracy and applicability of these experiments are discussed, and the results of control studies are reported.

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Mr. Robert E. Fletcher, Director of the Maico office in Cincinnati, assisted in taking the audiograms and provided facilities for these studies.

EFFECTS OF OXYGEN DEPRIVATION UPON THE STRUCTURE OF THE ORGAN OF CORTI

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AND
E. G. WEVER, Ph.D.
PRINCETON, N. J.

ONE OF the biological processes most resistant to oxygen deprivation is the generation of electrical potentials by the cochlea when this structure is stimulated by sound. But careful laboratory studies on animals have shown that under extreme anoxia deterioration in this process is obtained. In an earlier study [1] it was found that in the cat no changes in the potential take place when the amount of oxygen in the inspired air exceeds 4% but a mixture slightly more dilute than this causes an impairment of the recorded potentials. As respiration continues with this dilute mixture, the impairment progresses until a steady state is reached which may then be maintained for hours, as though the animal had adjusted to this low oxygen condition. A further decrease in the percentage of oxygen produces a further decrease in potential, rapid at first and again leveling off at a lower value. It is possible to continue this process until the heart degenerates to the point at which its action ceases. From this point onward the potentials steadily decline until they become too weak to be recorded.[2]

If normal air is introduced through the respirator after the animal has experienced a period of anoxia which has produced a condition of lowered but now stabilized potentials, these potentials will rise somewhat, indicating a recovery of function, but this rise never reaches the level existing prior to the state of anoxia. Some seemingly permanent deterioration has taken place, or at least no full recovery has been manifested during the few hours of breathing normal air in these experiments. Whether complete recovery could be obtained if this breathing of normal air were continued for weeks or months is not known, but there is no good reason to believe that this would occur.

A further test was made in the foregoing experiments to ascertain whether there was any differential change in sensitivity to 12 different frequencies over the range from 100 to 15,000 cycles. This was done by finding the magnitude of stimulus necessary to produce 1μ of response at the various frequencies. Such a

From the Princeton Psychological Laboratory. This research was supported in part by the Office of Naval Research, under Contract N6-onr-270, T. O. 3, Project NR 140-322. Permission is granted for reproduction, translation, publication, use, and disposal in whole or in part by or for the United States Government.

1. Wever, E. G.; Lawrence, M.; Hemphill, R. W., and Straut, C. B.: Effects of Oxygen Deprivation upon the Cochlear Potentials, Am. J. Physiol. **159**:199-208, 1949.

2. Wever, E. G.; Bray, C. W., and Lawrence, M.: The Nature of Cochlear Activity After Death, Ann. Otol. Rhin. & Laryng. **50**:317-329, 1941.

sensitivity curve was taken before and after the animal was exposed to a mixture of 0.5% oxygen for 30 minutes. If slight corrections were made for the further deterioration that occurred during the time it took to carry out the measurements, the second sensitivity curve appeared identical with the first, from which evidence it appears that the deterioration of cochlear potentials due to anoxia is independent of frequency.

The sensory structure dealt with in these experiments is, of course, the organ of Corti and, more specifically, as many lines of evidence indicate, the hair cells. The exact process involved in the conversion of mechanical energy into electrical energy is not clear, but there is little doubt that the potentials recorded from the vicinity of the cochlea originate in the hair cells as they are physically distorted by the acoustic vibrations.[3] In order to pursue the problem of how the lack of oxygen affects the cochlear potentials, fixing fluid was injected intravitally into some of the animals used in the anoxia study, and they were saved for subsequent histological examination.

RESULTS OF HISTOLOGICAL EXAMINATION

Of the animals used in the previous experiment, four (eight ears) were brought to final histological study. Three of these were fixed intravitally with Maximow's solution; the fourth, as the result of lowered oxygen, died before an injection could be given, but the animal was placed in fixing fluid immediately upon death. When the ears were finally sectioned and mounted, they were mapped by the graphic reconstruction method described by Guild.[4] This was done in order to locate the areas of degeneration and to provide a systematic picture of the condition of the ear.

These animals were in different states of anoxia when fixed, but the general picture of degeneration was much the same. Every ear showed along the extent of the cochlea a number of degrees of alteration that seemed to represent progressive stages in the degenerative process. They differed only in that the animals exposed to the severest anoxia showed extreme stages of degeneration.

The basal end of the cochlea always showed more serious deterioration than the apical end, but otherwise there was no fixed pattern of changes. The pattern was spotty; there were particular areas along the cochlea in which the organ of Corti showed worse degeneration than at places immediately above and below.

The stria vascularis also varied from almost normal to a condition of complete degeneration, and the state of this structure seemed to have no relation to the state of the organ of Corti in the immediate vicinity.

Of the eight ears studied, two were considerably degenerated and will be reported on shortly. The other six all presented the same picture. In order to show the distribution of degenerative changes along the length of the organ of Corti, an ear of one of the animals was selected and is represented in a series of photomicrographs.

This animal was exposed to a mixture of nitrogen and air with an oxygen content of only 0.5%. The first exposure of about 10 minutes dropped the recorded potentials nearly 15 db. After a period of air-breathing there was a second exposure to the same low oxygen mixture, and after another period of air-breathing, a third

3. For a brief review of this evidence, see Lempert, J.; Wever, E. G., and Lawrence, M.: The Cochleogram and Its Clinical Application, Arch. Otolaryng. **45**:61-67 (Jan.) 1947.

4. Guild, S. R.: A Graphic Reconstruction Method for the Study of the Organ of Corti, Anat. Rec. **22**:141-157, 1921.

exposure; all of these resulted in a total loss of potentials of 20 db. After about an hour of normal air-breathing, the 0.5% oxygen mixture was again administered. This caused the heart to fail badly and the total cochlear potential loss to reach 25 db. Epinephrine was injected into the heart, and in two minutes the pulse was strong and regular, and the potentials had recovered to a point 13 db. below the normal level, where they remained until the animal was perfused for the histological study.

Figure 1 portrays a section of the organ of Corti from near the apex of the cochlea where the least amount of degeneration is evident. Here the hair cells are clearly seen in their usual positions and are almost normal in form. Certain of the supporting cells, however, show degenerative changes. The Deiters cells are

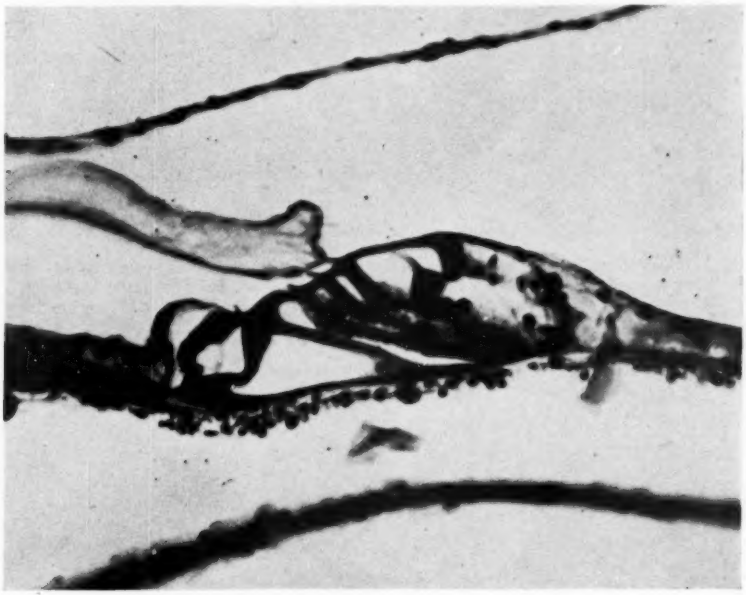

Fig. 1.—An early stage of anoxic degeneration. The supporting cells of Deiters have begun to shrivel up, losing their distinctive form and producing a hyalin exudate. Cat 308-764-R; section taken from the apical region (at a point 98% of the distance from the basal end); × 370.

shriveled, and the Hensen cells are undergoing dissolution. Other structures are essentially normal in appearance.

At another place in this ear the disintegrative process evidently had proceeded further, as shown in Figure 2. Here the external sulcus cells have been affected and are greatly swollen and have indistinct boundaries. Yet the hair cells still appear as distinct units and stain in a normal fashion.

The hair cells, after the remaining cells have lost their normal appearance, finally begin to be affected. Figure 3 presents this picture. Now the supporting cells have almost completely lost their shape and boundaries, while the hair cells have become shrunken and twisted. Some seem to have lost their contents with no semblance of a nucleus remaining. The shrinking apparently starts in the region of the Deiters cup. The lower end of the hair cell is reduced to an irregular point, while the

Fig. 2.—Further anoxic degeneration. Deiters cells have now almost completely lost their form and the cells of Claudius and Hensen have swollen and begun to disintegrate. Buckling of the basilar membrane and loss of the tectorial membrane are due to the histological treatment. Cat 308-764-R; section taken from the middle region (55% of the distance from the basal end); × 345.

Fig. 3.—As the degeneration progresses the hair cells become involved and also begin to shrivel up and lose their form. Cat 308-764-R; section from the apex (92% of the distance from the basal end); × 370.

upper end remains of normal width. Though the body of the Deiters cell becomes completely disintegrated, the phalangeal processes can occasionally be detected. These processes along with the rods of the pillar cells resist disintegration until the last stages, keeping the reticular membrane in place and holding the upper ends of the hair cells in their normal position.

Figure 4 shows a section of the organ of Corti near the basal end of the cochlea. Disintegration has proceeded to the stage where the Hensen and Claudius cells have deteriorated into strands detaching from the basilar membrane and seemingly beginning to disappear. The Deiters cells and hair cells are completely disorganized and beginning to fall into a formless mass.

Fig. 4.—Further anoxic degeneration in which the organ of Corti is beginning to lose its distinctive form, with all cells becoming shapeless and disintegrated. Cat 308-764-R; section from the basal region (34% of the distance from the basal end); × 345.

In certain regions the stria vascularis has been affected. The form of its disintegration is shown in Figure 5. The cells have broken up into fibroid strings, giving off a hyaline exudate that has washed out into the endolymph.

It might be expected that the regions of degeneration of the stria would be related to those in which the organ of Corti is affected, but no such relation was found. In the animal illustrated here, the area of disintegration of the stria extended from 2.6 to 5.4 mm. from its basal end, and thus comprised 14% of its total length. The organ of Corti was affected far more extensively than this, and no more seriously in this region than elsewhere.

Figure 6 represents a section of the organ of Corti in another animal which evidently suffered from anoxia of a particularly high degree. This animal was used in another experiment and was under a dosage of diallylbarbituric acid and curare and was maintained by artificial respiration. Through a fault in the respira-

tor, however, the animal deteriorated over a period of an hour or two to the point at which the cochlear potentials were reduced in both ears to only a trace. At this time the animal appeared normal in other respects, with a satisfactory heart beat.

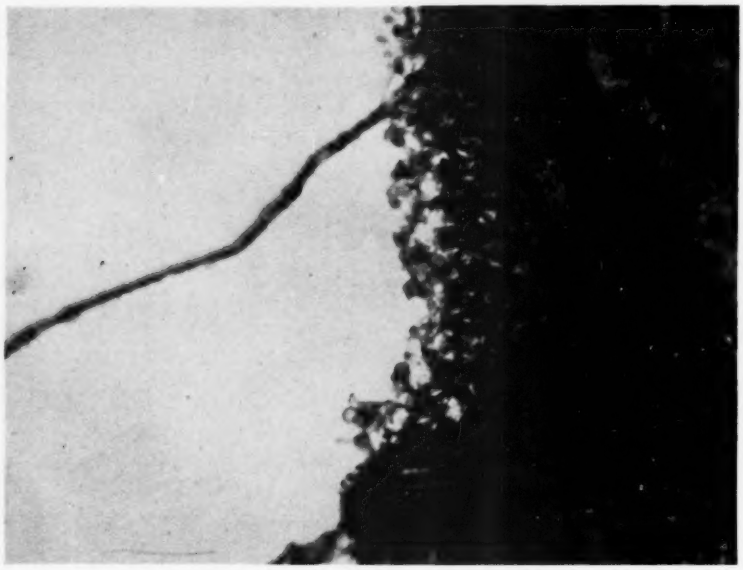

Fig. 5.—In certain unpredictable areas the stria vascularis undergoes disintegration as shown here. Cat 308-764-R; section from the basal region (15% of the distance from the basal end); × 175.

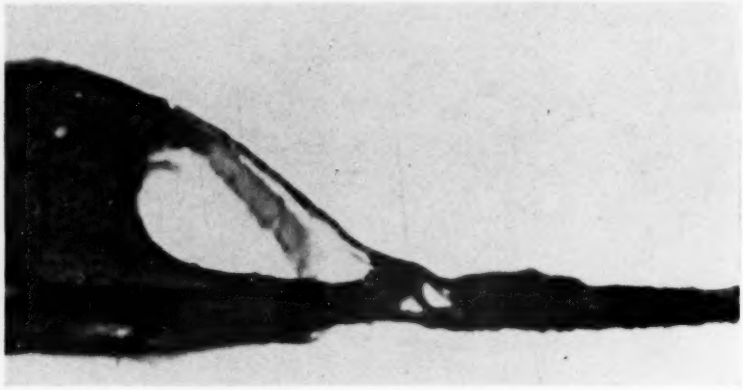

Fig. 6.—A section from the basal turn of an animal which became nearly deaf as the degeneration shown here took place. Cat 157-578-L; × 300.

It was promptly given an injection intravitally and sent through the histological process, with the results as shown.

In Figure 6, the organ of Corti has been reduced to a misshapen mass with only the arch recognizable, and Reissner's membrane has collapsed to cover the

entire structure. This condition prevails over the complete length of the organ of Corti.

Such gross disintegration can well account for the loss of cochlear potentials. This is probably the picture that extreme anoxia presents, but curiously we have not succeeded in producing it deliberately in the living animal. In all other animals of this study the extremes of anoxia that were necessary to produce such a deterioration of the potentials have always damaged the heart so seriously as to cause death.

COMMENT

It is of interest that when the organ of Corti is subjected to an abnormal environment of lowered oxygen, it is the supporting cells that first succumb, while the hair cells are well maintained up to the later stages of the degenerative process. In the ear first discussed here, almost the whole of the sensory structure was affected in some degree, yet along 85% of its course the hair cells were still discernible. How efficient in function these cells were is a further object of interest. It is unlikely that these cells, when as seriously affected as in Figure 4, could carry on their normal activities in response to sounds. The final impairment of cochlear potentials was to the extent of 13 db., or a loss of 78%. This figure may represent the over-all deterioration of function, but obviously it is not possible to ascertain the degree of function of any particular cell or region. It is noteworthy, however, that the hair cells in certain regions were very profoundly affected, yet the same degree of impairment of cochlear potentials was observed for all frequencies.

The changes of function occurring within the cochlea after the death of the animal present the same pattern of impairment as those changes resulting from anoxia. In the study of cochlear potentials after death it was found that three hours after an animal was killed by curare poisoning, the loss in cochlear potentials was 16 db. This is only 3 db. more than the final loss sustained by the first animal presented here, and less than the greatest loss this animal experienced before it was allowed to recover with breathing of normal air. It appears that the organ of Corti retains a good deal of its viability even three hours after cessation of the heart and would make a substantial recovery of function if oxygen were again provided. A more violent death than that caused by curare poisoning, brought about by clamping the trachea, produced a loss in potentials of 34 db. three hours after the heart stopped. This greater loss was due, no doubt, to the more rapid consumption of the oxygen remaining in the system by the greater muscular activity involved in this form of death. Apparently, nonpathological changes in the organ of Corti, seen upon histological examination, depend not only on the length of time elapsing between death and fixation but also on the nature of the death and the physiological state of the animal at the time of death.

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CLOSURE OF TYMPANIC MEMBRANE PERFORATIONS BY TRICHLOROACETIC ACID CAUTERIZATION

Report of Cases

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FOR MANY years physicians have been interested in the problem of how to close tympanic perforations, and numerous methods have been employed. The method to be considered here is also an old one, namely that of cauterization of the margins of the perforation with trichloroacetic acid, which was apparently first used by Okuneff in 1895.

Justification for this type of treatment seems to lie in the fact that following a perforation of some duration the epithelial outer layer of the tympanic membrane tends to grow over the margin of the perforation toward and sometimes into the middle ear. This leads to the formation of an epithelized hole or communication to the middle ear which then has no chance to close. If the epithelium could be discouraged from growing inward until the fibrous and mucous membrane layers could form a structure or bridge for this migration, one might expect to close the perforation. Figure 1 demonstrates this migration of squamous epithelium over the edge of the perforation.

With this in mind, 12 perforations were treated by destruction of their epithelial lining with trichloroacetic acid; in this series there was permanent closure of 10 perforations and failure of closure of 2 with subsequent rupture of one of the healed membranes.

The general technique used in this series was to apply destructive strengths of trichloroacetic acid, usually 50 to 75% solution, to the margins and surrounding edge of the perforation by means of a fine cotton-tipped metal applicator. On the first application, an attempt was made to abrade or rub the edges of the perforation —not simply to apply the acid. Under direct vision whitening of the edges was noted, and unless this whitening was observed a second application was made at the same time. Care was taken to be sure the surrounding area as well for several millimeters was touched in an effort to further retard the epithelial progression. Figure 2 shows the areas cauterized.

Appearance of the growing edge, of course, altered further treatment, but once a granulating edge was noted, weaker dilutions of acid were used and were applied as nearly as possible to the immediate area surrounding the margin. It is felt that these doses were not stimulating but rather retarding, since even with a 5 to 10% solution of acid whitening of the epithelium would occur. It was usually necessary to apply acid once or twice every 10 days, but if healing was obvious and rapid

Read before a meeting of the Philadelphia Laryngological Society, Philadelphia, April 3, 1951.

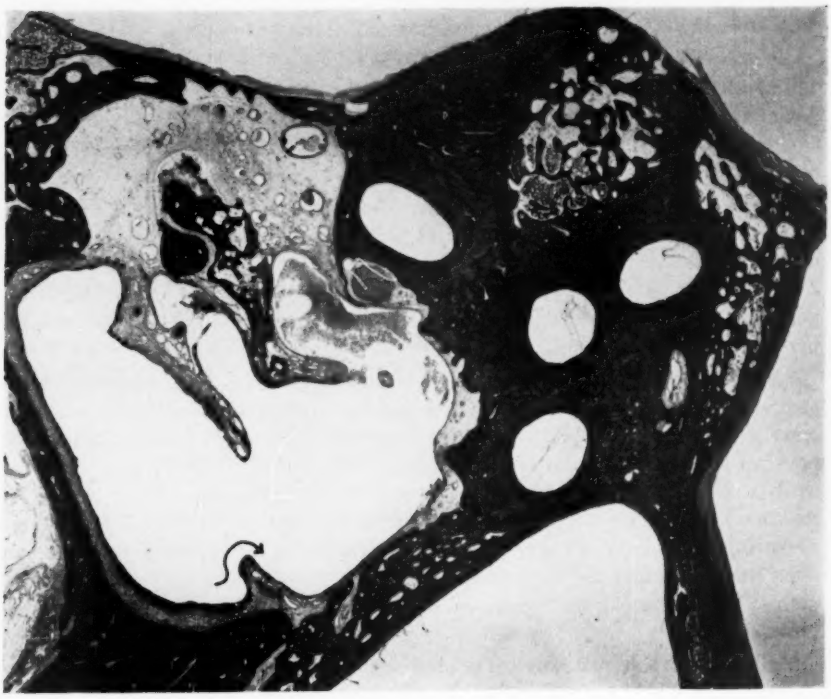

Fig. 1.—Migration of squamous epithelium over edge of perforation. (Used with permission of Dr. Stacy Guild.)

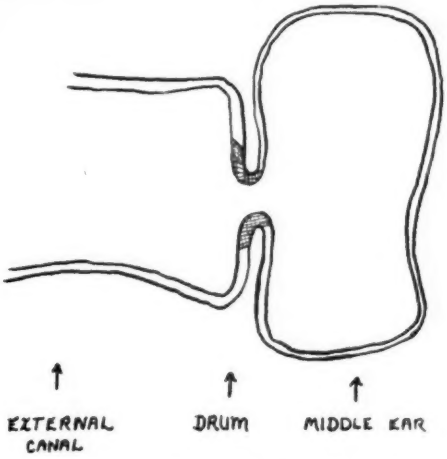

Fig. 2.—Cauterization of membrane. The shaded area indicates the extent of cauterization.

no application was made. Dunlap and Schuknecht [1] state that the epithelium can grow over the edge of the perforation in 4 to 7 days.

It was easy to determine a healing edge because of the characteristic pink rim which caught the illumination readily, giving it a luminous appearance. This was definitely different from the pale rolled edge of an inactive area. Frequently the two types of edge appeared in the same perforation, and cauterization of only a portion of the perforation was necessary.

It was frequently noted that an eschar formed around the perforation and could be removed to show a complete cast of the cauterized area with the perforation demonstrated—very much like the hole in a doughnut. It was felt that such casts should remain in place until activity was no longer apparent. Occasionally the perforation would close leaving the crust in place with a cast of the original perforation superimposed.

Although it is perhaps wise to use local anesthesia, for example, Bouin's solution (a solution of equal parts of cocaine, menthol, and phenol), before the first application, in this series no anesthetic was used. The pain is short-lasting and does not seem to bother the patient greatly. Furthermore, either the pain was more easily tolerated after the first treatment or it was actually less. Time needed for treatment is minimal when an anesthetic does not need to be considered. Before the treatment of a patient was begun, it was explained that patience and perseverance were necessary for an indefinite time but that because of the value to the patient of an intact drum membrane I felt that the treatment was well worth the effort.

The type of ear that was considered suitable for an attempt at closure of the membrane had a history of intermittent drainage; however, several times therapy was started during drainage if the history and physical findings showed that it was a case of recurrent drainage with intervening dry periods. These ears became dry before final closure was accomplished. No ear in which there was antral or attic disease was accepted for treatment, and in each case the perforation was of the membrana tensa. A patent Eustachian tube was another prerequisite.

All the patients who faithfully continued to come for treatment are included in this survey. There was one other in whom there was the beginning of closure who failed to return, as well as one in whom no closure was noted at the last visit.

It is interesting to note that after the healing of the perforation it is practically impossible to tell the site of the original perforation—in only one of these patients was the perforation closed with atrophic membrane, and this was in a patient 55 years of age who had a marginal perforation of long duration The shortest time interval for closure was 16 days, which occurred in a patient with intermittent otorrhea for two years with a small polyp extruding from a large posterior-inferior perforation. This time interval includes destruction of the polyp by the same method of cautery. The longest time interval necessary for closure was 119 days, which was needed by a patient approximately 35 years of age with a dry ear and pale tympanic membrane with a history of perforation for one and one-half years. The average time necessary for closure was seven weeks—49 days.

1. Dunlap, A. M., and Schuknecht, H. F.: Closure of Perforations of the Tympanic Membrane, Laryngoscope **57**:479-490 (July) 1947.

It was noted that some patients showed a classic loss of hearing for low tones; others a loss of hearing for a combination of high and low tones. This finding is consistent with the findings and opinion of Dr. Stacy Guild,[2] who feels that the impairment of thresholds by air conduction, with each of the conditions that cause conductive deafness, not only may differ in amount from slight to marked but also may be approximately equal for all tones, greater for low than for high tones, or greater for high than for low tones. In this series I noted that in every patient there was improvement and a return to an efficient hearing level. The one patient who did not show a return to such a level showed a significant and helpful increase in hearing acuity. Figure 3 and the table show graphically the average prehealing and posthealing audiograms.

It is realized in evaluating these figures of hearing gain that one must consider the factor of learning in the testing procedure which would make this gain more apparent than real; moreover, the ears were in varying stages of inflammation and

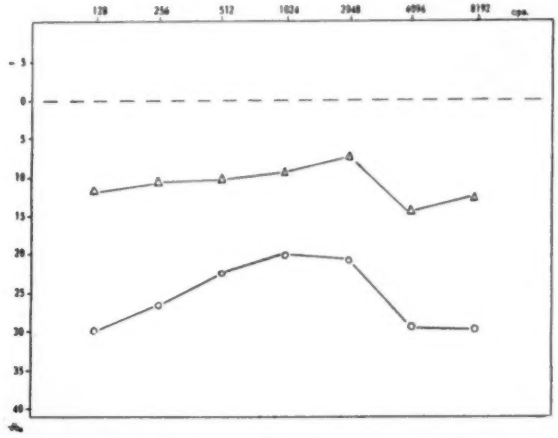

Fig. 3.—Average preclosure and postclosure audiograms. The line with triangles indicates postclosure and the line with circles preclosure audiograms.

infection when the initial audiogram was taken. Some patients had a combination of conductive and perceptive deafness. Nevertheless there is a remarkable similarity in the prehealing audiograms of the different patients. All factors considered, it appears that there is an appreciable hearing improvement. This is in accordance with the findings of Dunlap and Schuknecht,[1] who state that if the ossicular chain is intact hearing is almost certain to be improved.

There were two patients in whom the membrane failed to close completely. One patient had a perforation involving the entire membrana tensa with a portion of the malleus handle eroded. In four months of treatment approximately 20% closure was noted. The second patient had a posterior-inferior perforation and was treated for six and one-half months without any appreciable closure. This is the only case in which there was no tendency to close. One possible explanation was

2. Guild, S. R.: Interpretation of Hearing Tests with Special Reference to Conductive Deafness, J. A. M. A. **142**:466-469 (Feb. 18) 1950.

the avascularity of the membrane; anatomically this area has the poorest blood supply of any portion of the drum. In one patient 59 years of age who had a marginal perforation for over 15 years the thin membrane that closed the perforation was ruptured subsequent to healing. When the patient was last seen and treated, this rupture was beginning to heal.

It is obvious to us all what closure of a tympanic perforation can mean to a person who lives constantly under the threat of a discharging and increasingly serious condition in the ear; he can then consider his ear safe and relatively normal. Then such a person can go swimming without feeling relatively certain that very

Preclosure and Postclosure Audiograms

		Hearing in Decibels at Frequencies of						
Case		128	256	512	1024	2048	4096	8192
1	Preclosure	25	25	35	35	30	25	20
	Postclosure	5	5	5	5	0	5	5
2	Preclosure	25	25	20	20	10	70	75
	Postclosure	0	— 5	— 5	— 5	— 5	45	30
3	Preclosure	20	25	20	5	10	15	15
	Postclosure	10	10	10	0	—10	— 5	10
4	Preclosure	25	35	15	15	15	10	25
	Postclosure	5	10	5	5	5	10	5
5	Preclosure	25	15	15	15	15	25	20
	Postclosure	5	0	5	10	0	0	5
6	Preclosure	30	20	10	15	30	25	45
	Postclosure	10	5	5	5	20	10	35
7	Preclosure	20	15	0	5	0	15	15
	Postclosure	10	5	— 5	0	5	5	— 5
8	Preclosure	35	25	20	10	15	15	25
	Postclosure	15	20	20	15	15	15	..
9	Preclosure	50	45	55	50	50	50	55
	Postclosure	35	35	50	40	25	35	35
10	Preclosure	40	40	40	35	35	45	5
	Postclosure	25	25	15	20	20	25	10
11	Preclosure	25	25	15	20	20	25	55
	Did not close	..	..	..	..	..	..	..
12	Preclosure	35	30	25	25	15	25	40
	Did not close	..	..	..	..	..	..	..

shortly his ear will begin to drain again. An intact membrane may enable him to pass his physical examination for the military service; he may notice a definite increase in auditory acuity and consequent help in interpretation of the source of sound. Occasionally a patient will report a disappearance or improvement in tinnitus, i. e., the entrance of sound masks his tinnitus.

SUMMARY

In presenting these cases, I have tried to show that with perseverance the majority of tympanic membrane perforations in selected cases of chronic middle ear disease will close. It seems to be well worth the time and effort necessary to achieve closure, since the patient then has a relatively normally functioning ear and, with few exceptions, an improvement in hearing.

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FIBROUS DYSPLASIA OF FACIAL BONES AND PARANASAL SINUSES

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THE SUBJECT of this paper is the relationship of the paranasal sinuses and fibrous dysplasia. It is likely that all the paranasal sinuses may be involved by fibrous dysplasia, although, to my knowledge, there is no complete case report concerning the sphenoid sinus. Only Mayer [1] mentions the x-ray findings of the sphenoid sinus in a patient who apparently suffered from fibrous dysplasia. However, since the sphenoid sinus is not as easily accessible as the other sinuses, it is likely that it has not infrequently escaped clinical investigation. This, however, does not indicate that fibrous dysplasia necessarily originates in any of the sinuses. Several authors emphasize that the disease prefers the membrane bones of the face. The ethmoid sinus and the sphenoid sinus, however, develop in a bone which is preceded by cartilage. At present, it cannot be stated whether or not in fibrous dysplasia of the ethmoid sinus the disease originates in the ethmoid or invades this sinus secondarily.

In fibrous dysplasia the sinuses are supposed to be entirely filled with newly formed tissue. Pugh [2] and Windholz [3] stress that in fibrous dysplasia of the frontal, sphenoid, ethmoid, and maxillary bones there is deposition of dense, sclerotic bone, while in the vault of the skull the disease has more frequently a cyst-like appearance in the x-ray film. Cases of fibrous dysplasia of the sinuses are not very common. Among 67 cases of fibrous dysplasia of single bones reported by Schlumberger,[4] the maxilla was involved in 7 and the mandible in 2. In two cases the disease involved the frontal bone. For this reason a report of two cases of fibrous dysplasia, of the maxilla and the frontal bone, respectively, seems to be warranted.

CASE 1.—Z. E., a white man aged 53, noticed tearing of the left eye and slightly blurred vision four months before he consulted me. Proptosis of the left eye developed, and the skin of the left cheek became red (Fig. 1). No double vision and no nasal troubles were noticed. There was no contagious disease and no trauma.

On Aug. 3, 1946, the Kahn reaction of the blood was negative. There were paresthesias of the left cheek and proptosis of the left eye, and the skin of the inner angle of and below the left eye was red and swollen. The left eye was slightly higher than the right. There was no tenderness of the left supraorbital nerve, and palpation did not reveal a tumor in the orbit.

From the Department of Otolaryngology, University of Illinois College of Medicine.

1. Mayer, E. G.: Die Röntgendiagnose der Erkrankungen der Schädelbasis, Wien. klin. Wchnschr. **45**:1022, 1932.

2. Pugh, D. G.: Fibrous Dysplasia of the Skull: Probable Explanation for Leontiasis Ossea, Radiology **44**:548, 1945.

3. Windholz, F.: Cranial Manifestations of Fibrous Dysplasia of the Bone: Their Relation to Leontiasis Ossea and to Simple Bone Cysts of the Vault, Am. J. Roentgenol. **58**:51, 1947.

4. Schlumberger, H. G.: Fibrous Dysplasia of Single Bones (Monostotic Fibrous Dysplasia), Mil. Surgeon **99**:504, 1946.

The dilation of the external orifice of the nose hurt. There was deviation of the septum to the right, but there was no tumor or pus. The mucosa of the turbinates was swollen. There was no diastema of teeth, and only the molars were absent on both sides. X-ray films showed no evidence of productive or destructive bone change involving the cranial vault or the bones of the base of the skull. The pineal body was calcified and appeared to lie in a normal position. No other intracranial calcifications were seen. The mastoids were composed entirely of diploetic bone. The sella turcica appeared normal. There was dense clouding of the left frontal sinus, the bony margins of which were sclerotic and eburnated. The right frontal sinus appeared essentially clear. The left ethmoid sinus was densely cloudy, and the right ethmoid sinus was clear. There was clouding of both antra, which was particularly dense on the left, where the air space was entirely obliterated. The bony margins of the antra appeared intact. The sphenoid sinuses were essentially clear. There was a bony bridge crossing the superior

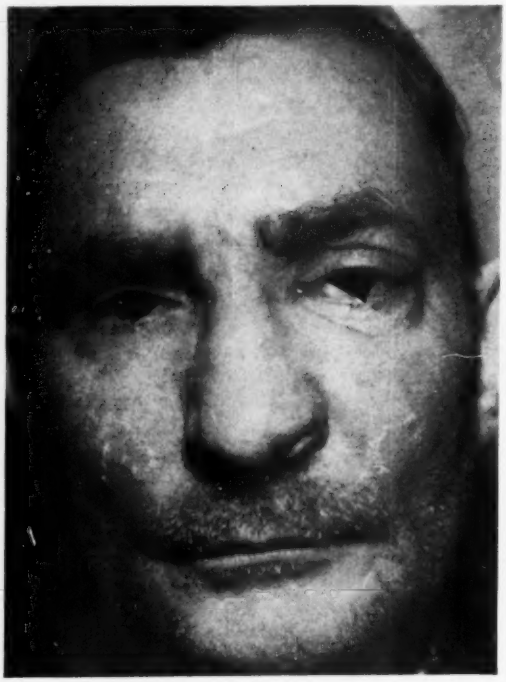

Fig. 1 (Case 1).—Photograph of patient on Aug. 30, 1946.

nasal meatus on the left. The optic canals were symmetrically formed and showed no atrophy or erosions. The posterior ethmoid cells were clear bilaterally, and the bony margins of the orbits were intact. An eye examination on Aug. 8, 1946, revealed marked proptosis of the left eye. Movements of the eye were limited in all directions, with the greatest limitation laterally. The left pupil was miotic but reacted to light. The lower bulbar conjunctiva showed telangiectasia. The left fundus revealed dilated veins and obscured disk margins. The tension and vision were normal. The left visual field was constricted in all directions; the right was normal.

On Aug. 17, a Denker operation was performed on the left maxillary sinus. The periosteum of the fossa canina was not adherent, and the cortex had a normal appearance. The bone of the apertura piriformis was removed, and a cavity 1 cm. deep was made into the bone. There was no lumen of the maxillary sinus. Apparently the entire left maxillary sinus was filled with cancellous, hyperemic bone, which had fused with the compact bone of the anterior,

mesial, lateral, and inferior walls of the sinus. More bone was removed, and the cavity was covered with the mucosa of the inferior nasal meatus. On Sept. 3, the incision in the mouth was closed. Until October, 1948, the condition remained unchanged.

Microscopic examination showed the bone to be cancellous, with remnants of cortical bone covered by a thickened periosteum. The bone trabeculae consisted of woven bone; there were no mosaic structures. The margins of the trabeculae were aplastic; occasionally there was some osteoid. Between the trabeculae there were firm or loose connective tissue and chronic inflammatory tissue, which, however, did not exert any influence upon the bone. The cells of the chronic inflammatory process were lymphocytes and plasma cells. In addition, there were large round or polygonal cells with a central pyknotic nucleus scattered within the loose connective tissue.

In this case there was a fibrous hyperplasia of the left maxilla. In the x-ray films there was seen to be, in addition, dense clouding of the left frontal and left anterior ethmoid sinuses. It is likely that the latter was likewise involved by fibrous dysplasia, inasmuch as there was no clinical evidence of a chronic infection. The bony bridge in the upper portion of the left nostril cannot be explained. The clinical symptoms were tearing, proptosis, displacement of the left eye, redness of the skin of the left cheek, and paresthesias of the left cheek. At operation the left maxillary sinus was found to be filled with cancellous bone, which in the x-ray films seemed to be sclerotic bone. Certainly these conditions did not develop in a period of four months; rather, the disease became manifest in the last four months.

At operation a piece of newly formed bone was removed; yet the defect was very rapidly filled with new bone. This is apparently typical of this disease. In a case of Cooke and Powers,[5] that of a youth aged 18 suffering from fibrous dysplasia of the maxillary sinus, three operations were performed on the sinus, with intervals of six months and two months respectively; yet at all these operations the sinus was found to be filled with cancellous bone. This is an interesting finding because, in general, the growth potential of the newly formed tissue in fibrous dysplasia is supposed to be low.

CASE 2.—J. Q., a white man aged 45, had never had injuries or any contagious disease. When he was 16, he noticed a swelling in the mesial portion of the floor of the right frontal sinus above the inner angle of the right eye. Later the right supraorbital arch became prominent and the right eye became gradually displaced. He never had severe headaches or double vision. Vision was good. He believed that in the last 10 years the tumor had grown faster than previously.

Examination on March 9, 1950, revealed a large bony tumor which involved the right supraorbital arch and extended over the glabella to the center of the left supraorbital arch. Laterally, the tumor was separated from the right parietal bone by a distinct line which extended from the outer angle of the right eye upward and backward for a distance of about 6 cm. The tumor extended into the frontal squama; however, in this area the border line was not distinct. Above the outer angle of the right eye the tumor extended upward into the frontal squama almost to the hairline; the extension into the frontal squama decreased gradually toward the glabella and the left supraorbital arch. The floor of the right frontal sinus was prominent, and there was a round tumor in its mesial portion, which had a broad basis and a bony consistency and bulged toward the internal palpebral ligament (Fig. 2*A* and *B*). There was no defect in the floor of the sinus. The ridge of the nose was normal. There was also a tumor in the mesial portion of the floor of the left frontal sinus, which was less prominent than the tumor on the right side. The skin over the tumors was normal and showed normal rugae except in the midline, where the skin was perfectly smooth. There was no tenderness. The right eye was displaced downward about 5 mm. There was slight enophthal-

5. Cooke, S. L., and Powers, W. H.: Monostotic Fibrous Dysplasia: Report of Two Cases, Arch. Otolaryng. **50**:319 (Sept.) 1949.

mos on the right side. Otherwise the eyes were normal. The nose was slightly pushed to the left side. There was pronounced septum deviation to the left. The agger nasi on the right side bulged markedly into the nostril.

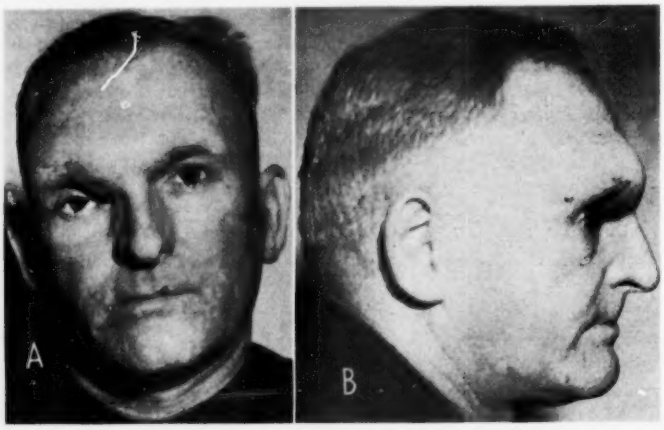

Fig. 2 (Case 2).—*A*, front view of patient; *B*, profile.

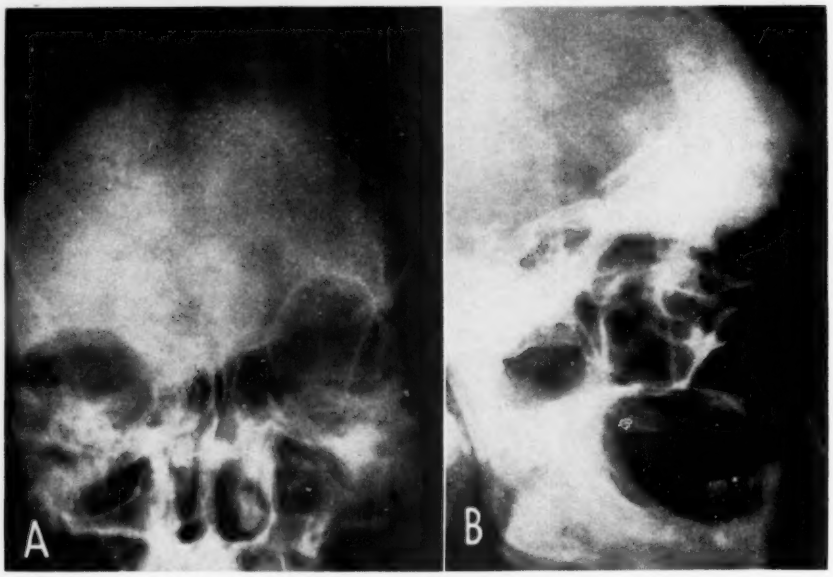

Fig. 3 (Case 2).—Roentgenograms of patient.

The x-ray films (Fig. 3) revealed in the region of the anterior portion of the frontal bone a large accumulation of sclerotic, dense, poorly or completely calcified bone. This tissue had completely replaced the right frontal sinus and had encroached on the left frontal sinus. The lesion was most marked in the right supraorbital arch and the zygomatic process of the right orbit. It did not cross the zygomatic frontal suture. The right anterior ethmoid cells were obliterated. The right supraorbital plate was dense and sclerotic with a fuzzy, irregular

ground-glass appearance in areas. In the region of the former right frontal sinus there were alternate clear-cut and fuzzy translucent areas of ground-glass appearance in which there were irregular, poorly ossified areas. The inner table of the frontal bone was indistinctly defined, but the outer surface was smooth. The lesion seemed to involve the cribriform plate but did not extend into the planum sphenoidale. There was a sharp boundary to this lesion. There were calcifications to be noted in the Pacchionian bodies in the parietal region. The rest of the skeleton did not show signs of fibrous dysplasia.

On March 18, a Killian incision was made on the right side. The floor of the right frontal sinus bulged considerably. The bone was apparently normal. The entire frontal sinus was filled with cancellous, moderately hemorrhagic bone. A piece of bone was removed.

The blood contained phosphorus, 3.5 mg. per 100 cc.; calcium, 8.8 mg.; alkaline phosphatase, 6 units, and acid phosphatase, 1 unit, indicating a normal condition.

The microscopic examination (Fig. 4) established that the specimen consisted throughout of cancellous bone, which extended up to the periosteum. There was no definite layer of compact bone. The bone trabeculae were formed of woven bone. There were marked osteogenesis and osteoclasis, the two processes being approximately in balance. The marrow spaces were filled with immature and mature connective tissue. In each marrow space there were

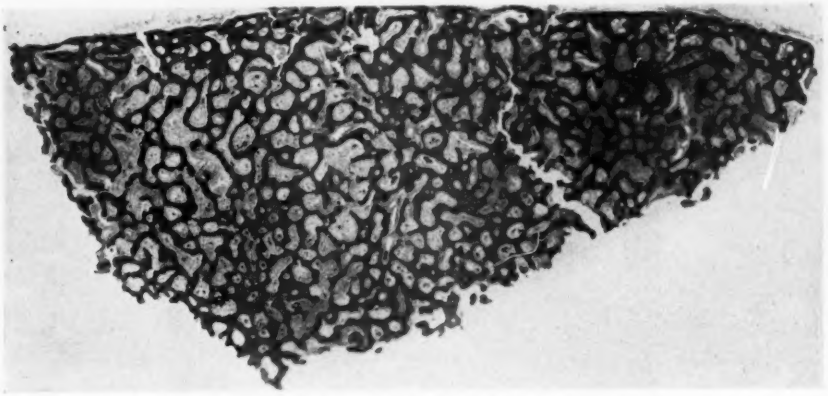

Fig. 4 (Case 2).—Section showing only cancellous bone and a few remnants of a substantia corticalis. The marrow spaces contain connective tissue and blood vessels; the process is almost exclusively osteogenesis.

capillaries or large, ramified blood vessels, which were empty and the walls of which consisted of endothelium only. Sometimes osteoclasts were found in the connective tissue. There were no signs of inflammation. There was approximately the same amount of bone and of connective tissue.

In Case 2 the area of the right frontal and ethmoid sinuses was replaced by newly formed, cancellous bone, a finding similar to that in Case 1. The symptoms were tumor formation at the forehead, the inner angle of the eye, and the supraorbital arch; displacement of the eye, and bulging of the roof of the orbit toward the anterior cranial fossa. The fibrous dysplasia had apparently encroached on the left frontal sinus. This case proves that fibrous dysplasia of the facial bones is not strictly unilateral.

In both Case 1 and Case 2 the surgical and the x-ray findings seemed to indicate that the respective sinuses had been entirely obliterated by newly formed cancellous bone. This is the usual interpretation of the findings but not the only possible explanation. The paranasal sinuses expand very slowly in the first six to seven years of life and reach their full size at approximately the age of 12 to 14 years

(Peter [6]). At birth the frontal sinus is not developed at all and the maxillary sinus is rudimentary. Since the onset of fibrous dysplasia is supposed to date back to childhood, it is evident that fibrous dysplasia must interfere if it is localized in the area of the paranasal sinuses. The following report of a case is thought to prove this conclusion.

CASE 3.—N. H., a white girl aged 7 years, was admitted to the hospital. About three years previously the parents first noticed that the child's nose was becoming wider between the eyes. There was no headache or visual disturbance. The nose continued to widen, and about one year after the onset the left eye began to be pushed down. Since then the condition had been progressive, without causing pain, visual difficulty, or illness of any kind.

Fig. 5 (Case 3).—Photograph of patient.

On admission on May 19, 1947, the girl was found to be well developed and the Wassermann reaction of the blood was negative. The hemoglobin content was 12.5 gm. per 100 cc.; the erythrocyte count, 4,170,000, and the leucocyte count, 7,500. The lower quarter of the forehead bulged anteriorly, and the left eyebrow had shifted laterally (Fig. 5). The bridge of the nose was twice its normal width, the enlargement appearing mostly on the left side. The superior margin of the left bony orbit was depressed 6 to 8 mm. below the eyebrow level. The left eyeball was displaced to an inferior, lateral, and anterior position. There was a slight exophthalmos. The inferior margin of the left orbit was 6 to 8 mm. lower than that of the right. The left nasal bone was markedly enlarged, causing the inner canthus of the left eye to be displaced laterally 8 mm. from a symmetrical position. The tip of the nose was slightly deviated to the left; the left corner of the mouth was depressed, and the left cheek was lower and less full than the right.

6. Peter, K.: Vergleichende Anatomie und Entwicklungsgeschichte der Nase und ihrer Nebenhöhlen, in Denker, A., and Kahler, O.: Handbuch der Hals- Nasen- Ohren-Heilkunde, Berlin, Julius Springer, 1925, vol. 1, p. 95.

The left pupil was dilated and fixed to light, with a diameter of 7 mm. Movements of the eyes were limited in all directions.

The nasal septum was deviated to the right, and the right nostril was normal. On the left side the inferior turbinate was normal. The middle meatus was occupied by a bony bulla, covered with normal mucosa, and pushing the septum to the right. There was no pus. Roentgenograms (Fig. 6) showed a small frontal sinus on the right side. On the left side, the upper portion of the nostril was widened and hazy; the ethmoid sinus was hazy, and the cribriform plate was distinguishable. There were only remnants of the lamina papyracea. There was no frontal sinus on the left side, and the superior margin of the orbit was absent. The roof of the left orbit bulged markedly toward the anterior cranial fossa. The roof of the left ethmoid sinus bulged toward the anterior cranial fossa, as well as toward the cribriform plate. The left maxillary sinus was small, cloudy, and displaced downward. The floor of the orbit seemed to bulge into the maxillary sinus. The sphenoid sinus was small and normal. There were no other changes in the skull.

On May 20, the bony bulla in the left middle meatus was opened. No fluid escaped. A sense of doughy resistance was received on palpation of the posterior ethmoid region. A biopsy specimen taken from the posterior ethmoid sinus did not permit definite diagnosis. Meningioma or fibroma was considered. On May 24, an arcuate incision was made above the left eye. The

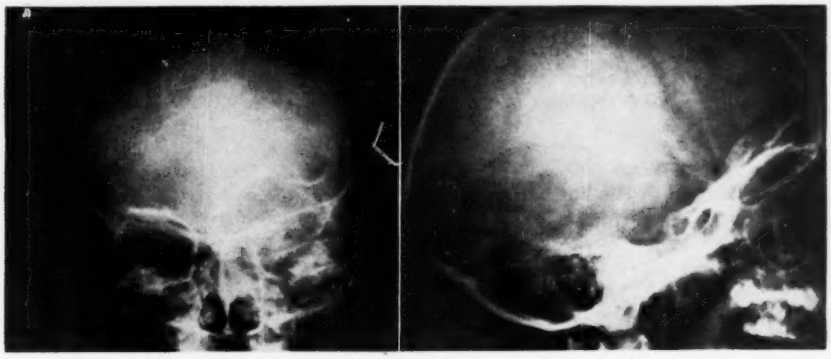

Fig. 6 (Case 3).—Roentgenogram taken prior to operation. Note the absence of frontal sinus, the bulging of the roof of the orbit on the left side, and the cloudy ethmoid sinus on the left side.

superior margin of the orbit and the frontal squama were exposed. The bone was discolored and soft, and there was a perforation with irregular boundaries and filled with blood coagula. The bone was removed with forceps. Immediately a tumor of yellow color and with the consistency of cheese was exposed. In the tumor there were several hemorrhages. The tumor was gritty and could not be removed in one piece. Fragments were removed until a large cavity resulted, bounded above by the roof of the orbit bulging into the anterior cranial fossa, laterally by the normal squama frontalis, and below by the normal periosteum of the orbit, the posterior portion of which was covered by a thin plate of apparently newly formed bone. Mesially the lamina papyracea was absent, and the tumor occupied the ethmoid area. The tumor had extended through the os nasale and the frontal process of the maxilla toward the skin; however, it did not involve the skin. Occipitally the tumor extended toward the sphenoid sinus, the bone of which was smooth and soft. The sphenoid sinus was not opened. Mesially it extended toward the nasal septum but did not reach the cribriform plate or the dura, nor did it reach the foramen opticum and the superior orbital fissure. The cavity was filled with iodoform gauze, and drainage through the nose was established. Uneventful recovery ensued.

The microscopic examination showed dense fibrous stroma composed of interlacing bonds of fusiform, heavy vesicular nuclei, which showed a tendency toward whorl formation, and moderately abundant acidophilic cytoplasm, which was poorly demarcated. There were sharply circumscribed, concentrically laminated basophilic bodies. There were, in addition, small irregular

foci of ossification and calcium deposition. These foci were outlined in some instances by homogeneous eosinophilic material, which compressed the surrounding stroma and gave the impression of expanding foci of ossification. The diagnosis was meningioma.

On Oct. 6, the exophthalmos had decreased but the displacement of the eye had not changed. In the inner angle of the left eye there was a tumor the size of a cherry, of bony consistency and covered by normal skin. The tumor extended toward the glabella. The middle meatus of the left nostril was filled with small polypi. The left nasofrontal duct was not patent. X-ray films showed a large translucent space in the area of the former tumor surrounded by a thick shell of bone. On Nov. 8, a second operation was performed. The tumor of the inner angle of the eye was found immediately under the skin and consisted of connective tissue covered by newly formed bone. The ethmoid sinus was filled with mucus and a moderate amount of gritty tumor tissue. The tumor extended under the skin along the back of the nose to the other side, covered by a thin layer of newly formed bone. Toward the nasal cavity the ethmoid sinus was sealed off by newly formed bone; toward the cavity in the frontal bone and the orbit it was sealed off by connective-tissue scars. The tumor was removed from the ethmoid cavity, but, owing to the extension and the gritty consistency, radical curettage was not possible. A free communication was created between the ethmoid cavity, on the one hand, and the nasal cavity and the cavity in the frontal squama, on the other. The cavity in the frontal squama did not contain tumor tissue and was a little smaller than at the first operation because of the soft tissue which bulged into the lateral portion. Uneventful recovery ensued.

The microscopic examination showed that the tissue of the supraorbital region consisted of fragments of bone and one piece of fibrous tissue. In a section of one fragment there was dense collagenous fibrous tissue with a few spicules of calcified material. A section of the bony tissue revealed trabeculated bone. Most of the trabecular surfaces were lined with osteoblasts and showed a bit of osteoid. The spaces were filled with a fibrous type of tissue containing blood vessels and areas of hemorrhage. In some areas fusiform cells were rather dense and rested in pink material. The latter fragment presented one curved surface, formed of moderately cellular connective tissue in which there was evidence of considerable bone resorption. The trabeculae of the adjacent bone were irregular in contour and architectural pattern and had an appearance consistent with fibrous dysplasia. The diagnosis was scar tissue and irregular cancellous bone consistent with ossifying fibroma.

In the fragments of the frontal region and the ethmoid region there were masses of fusiform cells with reddish cytoplasm and fusiform, bluish-purple nuclei, having a tendency toward whorl formation and growing around oval, deep-red bodies among fragments of bone. There was definite destruction of bone by the tumor process in the ethmoid area.

The obvious ossifying character of the lesion as seen in this specimen, in contrast to the specimens examined earlier, as well as its extension and progression within bone, made the diagnosis of meningioma less tenable. The diagnosis was ossifying fibroma with considerable growth potential.

Until April, 1949, no changes had been noticed. The child felt well and grew up normally.

When the girl was admitted to the hospital, she was 7 years old. According to the history, the disease was noticed first when she was 4 years old. At that age an actual frontal sinus has not developed. Since pneumatic spaces do not develop in pathological bone,[7] it is a fair conclusion that in the area involved by fibrous dysplasia a normal frontal sinus probably never will develop. It is likely that this girl, when she becomes mature, will present a condition similar to that in Case 2; yet it would be erroneous to state that in this patient the frontal sinus has been obliterated by newly formed tissue owing to fibrous dysplasia. In this patient there never was a frontal sinus, because fibrous dysplasia dated back to a time when the frontal sinus was not developed. It is likely that the same conclusion applies in Case 2. The patient was 45 years old on admission. He showed a photograph of

7. In a case of self-limited osteomyelitis of the frontal squama in a young child I observed the development of a normal frontal sinus on the involved side after successful removal of the bony sequestrum.

himself which proved that at the age of 16 the disease was almost as far advanced as at the age of 45. Considering the slow growth of fibrous dysplasia, it is probable that the onset of the disease occurred at an age at which the frontal sinus was not completely developed or not developed at all.

In general, the following conclusion can be drawn: Fibrous dysplasia may cause an obliteration of paranasal sinuses in some persons; in others, however, it prevents the normal development of the sinuses by producing a newly formed tissue which occupies the spaces destined for the paranasal sinuses. This concept explains the finding that usually the involved sinus is obliterated in its entirety, even in young persons. This likewise explains the fact that a radical removal of the newly formed tissue in fibrous dysplasia of paranasal sinuses, as a rule, is not possible unless the facial bone harboring the involved sinus is removed to a great extent—an operation which would not be justified.

The differentiation of failure of the sinus to develop, on one hand, and secondary obliteration of the sinus, on the other, is not difficult if the rest of the sinus is seen on the x-ray film. However, the differentiation is next to impossible if no trace of a sinus lumen is to be found. In such instances only the history may indicate whether or not the onset of fibrous dysplasia preceded the development of the sinus.

The x-ray findings are entirely different when a bone next to a sinus becomes involved by fibrous dysplasia. Two cases of fibrous dysplasia of the malar bone are reported briefly, since an exhaustive report was published from another point of view.[8] In one case the diagnosis was definite; in the other, likely.

CASE 4.—J. D., a white man aged 29, had had enlargement of the right cheekbone since birth. He had not noticed conspicuous growth of the swelling, but it increased in size in proportion to the body growth. There was no discomfort until two years before he consulted me, when he noticed that the right ear canal had become blocked. At present he is practically deaf in the right ear.

At examination, on April 20, 1945, the right malar bone was found to be replaced by a bone tumor the size of a plum. The tumor was covered by normal skin and extended to the inferior margin of the right orbit. The right eye was in normal position. The nasal findings were normal, and the right auditory canal was filled with exostoses. There was a small amount of pus between the exostoses. Hearing was markedly diminished on the right side. An x-ray film showed a pronounced thickening of the right malar bone. The bone at this site was not eburnated; it consisted, rather, of spongious bone with marrow spaces. There was a marked expansion of the right maxillary sinus into the malar bone. A similar bulging was noticed of the right sphenoid sinus, which extended deeply into the basilar bone.

At the operation, on May 8, 1945, the exostoses were removed from the auditory meatus. The tumor of the malar bone bulged slightly into the canal; it consisted of spongy bone, rich in blood vessels. A portion of the tumor was removed.

The microscopic examination showed the exostoses and the tumor to have essentially the same structure. They consisted of a network of bone trabeculae and connective tissue. The bone was a primitive, woven bone and contained a large amount of osteocytes. There was no lamellar bone. The trabeculae were covered with osteoblasts. There were frequent Howship lacunae, which also were covered by osteoblasts, while osteoclasts were found within the connective tissue. Many trabeculae were covered by a layer of osteoid. Between the trabeculae there was connective tissue rich in nuclei and blood vessels. In some sections there was metaplastic ossification within the connective tissue.

An uneventful recovery ensued, but soon the tumor bulged into the ear canal again. The calcium content of the blood serum was 11.3 mg. and the phosphorus content 3.8 mg. per 100 cc.

8. Brunner, H.; Spiesman, I. G., and Theobald, J. J.: Pneumosinus Dilatans, Ann. Otol. Rhin. & Laryng. **54**:763, 1945.

CASE 5.—V. E. B., a white man aged 35, had had enlargement of the right cheek since birth. The enlargement grew gradually, corresponding to the growth of the body. He had no pain and presented no history of fracture. Since childhood he had noticed tumors on the mucosa of the right cheek, on the right half of the lower lip, and on the right side of the tongue. There were no other tumors of the body. His hearing was diminished in the right ear. On June 6, 1927, most of the zygoma was removed surgically. Microscopic examination revealed cancellous bone and an abundance of hematopoietic tissue in the marrow spaces. The swelling recurred soon.

On May 28, 1945, a considerable enlargement of the right malar bone was noticed. The right ear canal was obliterated by exostoses. There were thickening of the inferior margin of the right orbit and enlargement of the right cheek. The alveolar process of the right maxilla was thickened and contained the premolar and two molars, all of which were covered by crowns. On the right side of the lower lip there was a tumor the size of a cherry stone. There were several tumors of similar size in the mucosa of the right cheek. All of these had a soft consistency, were covered by normal mucosa, were movable, and did not produce any symptoms. There was elephantiasis of the right half of the tongue. The nose was normal. There were 10 mg. of calcium and 3.2 mg. of phosphorus per 100 cc. of blood. Roentgenograms showed that the tumor consisted of a spongious bone with fine pores and involved particularly the body of the malar bone. There was no sclerosis of bone. The right maxillary sinus was definitely larger than the left and presented a malar recess extending toward the tumor and an alveolar recess extending toward the thickened alveolar process. There were no distinct boundaries between the lumen of the sinus and the tumor, since the tumor apparently extended into the maxillary sinus. The frontal sinuses were large. The right ethmoid sinus was hazy. The sphenoid sinus and the sella turcica were normal. There was a diminished pneumatization of the right temporal bone.

In both Case 4 and Case 5 the diagnosis was primarily osteitis fibrosa. According to the modern terminology, the diagnosis was changed to fibrous dysplasia. In Case 5 the diagnosis was not proved microscopically, but the clinical course and comparison with Case 4 render the diagnosis most likely. In both cases the maxillary sinus appeared to be dilated rather than obliterated according to the x-ray films, which presented the findings of pneumosinus dilatans. It is, however, questionable whether or not there was an actual dilatation of the maxillary sinuses. The findings in the ear canals suggest another interpretation of the roentgenograms. In both cases the ear canals were narrow owing to exostoses and to fibrous hyperplasia of the malar bone. It is likely that in fibrous dysplasia of the malar bone there is encroachment upon the maxillary sinus as well as upon the ear canals. If, however, the tissue formed by fibrous dysplasia is cancellous bone with poorly calcified trabeculae, the tumor bulging into the maxillary sinus does not cast a shadow on the x-ray film. Thus, the impression of a dilatation rather than an obliteration of the sinus is created.

There are, in addition, other findings in Cases 4 and 5 which are noteworthy. In both cases the disease extended into the malar process of the temporal bone; in other words, it was not strictly monostotic. This is apparently the rule rather than the exception. In Case 1 the frontal and the ethmoid bones were involved in addition to the superior maxilla; in Cases 2 and 3 the ethmoid bone was involved in addition to the frontal bone; in Case 4 the temporal bone was involved in addition to the malar bone; and in Case 5 the malar bone and the malar process of the temporal bone and the alveolar process of the superior maxilla were involved. Although it is not certain that in all of these bones exactly the same microscopic changes would have been discovered, the following conclusions can be drawn from these findings:

Fibrous dysplasia of the facial bones is not strictly monostotic, although it may be localized and predominantly unilateral; as a rule, several facial bones are involved, one bone presenting the maximum of changes. In some cases the disease crosses the midline and extends to the other side.

In Case 5 fibrous dysplasia of the malar bone was associated with tumors of the oral mucosa and of the tongue which were noticed on the same side as the fibrous dysplasia. Unfortunately, the patient did not permit the removal of one of these tumors. For this reason, an exact diagnosis is not possible, but it is likely that the tumors were fibromas. It seems that the association of fibrous dysplasia with tumors of the oral mucosa and of the tongue has not been observed at yet. This syndrome may throw some light upon the relation between fibrous dysplasia, on the one hand, and neurofibromatosis, on the other.

MICROSCOPIC OBSERVATIONS

In Cases 2 and 3 the same area of the skull was involved; yet the microscopic observations were different. In Case 2 (and also in Cases 1 and 4) the newly formed tissue consisted of a large amount of bone trabeculae and of a myelofibrosis rich in blood vessels. The bone trabeculae were formed by woven bone and replaced the substantia corticalis to a great extent. There were no mosaic structures. In Case 2 osteogenesis and osteoclasis were marked; in Case 1 they were not marked. The periosteum was normal in Case 2 and thickened in Case 1. In Case 1 there was, in addition, a chronic inflammation of the connective tissue.

In Case 3 there was a large amount of connective tissue, the nuclei of which showed a tendency toward whorl formation. Within the connective tissue concentrically laminated basophilic bodies, calcium, and a small amount of trabeculated bone were found. The first diagnosis was meningioma, which was changed later to ossifying fibroma with considerable growth potential. It should be noticed that the pathologist used the terms "fibrous dysplasia" and "ossifying fibroma" interchangeably.

Tumors of this type are noticed in the jaws. Phemister and Grimson [9] have called them "fibrous osteomas" while Hara [10] has reported on three patients, two of whom were observed by me, under the heading "ossifying fibroma." In the frontal area these tumors are less common. Benjamins [11] has given an excellent report of such a case and has reviewed six similar cases from the literature. The tumors were classified as osteoidoma, fibro-osteoma, sarcoma, osteoma, endothelioma psammosum, and osteoid fibroma with atypical calcification. Benjamins believes that these tumors originate from the periosteum of the roof of the orbit and that they do not produce mature bone but only osteoid, which is associated with a deposition

9. Phemister, D. B., and Grimson, K. S.: Fibrous Osteoma of the Jaws, Ann. Surg. **105**:564, 1937.

10. Hara, H. J.: Ossifying Fibroma of the Superior Maxilla, Arch. Otolaryng. **40**:180 (Sept.) 1944.

11. Benjamins, C. E.: Das Osteoid-Fibrom mit atypischer Verkalkung im sinus frontalis, Acta oto-laryng. **26**:26, 1938.

of frank calcium salts. Recently Woodruff [12] and Fry and DeLong [13] have reported similar cases under the heading "ossifying fibroma" and "psammoma" respectively.

Although the microscopic observations in Cases 1, 2, and 4, on one hand, and Case 3, on the other, differ in many respects, some authors (Lichtenstein,[14] Lichtenstein and Jaffe,[15] and Schlumberger [16]) believe that both groups of observations indicate simply variants of fibrous dysplasia. I do not believe that this question can be answered definitely. It should, however, be noted that a transition of one type into the other is not proved; each type has its specific characteristics from onset and adheres to them until adult life.

SUMMARY AND CONCLUSIONS

In many instances fibrous dysplasia of the skull prevents the development of the paranasal sinuses by producing pathological tissue which takes the place destined for the respective sinuses.

It is not proved that fibrous dysplasia causes actual obliteration of a normal paranasal sinus, but it is proved that actual obliteration of a sinus does occur, for instance, in osteitis deformans.[17]

Two cases of fibrous dysplasia of the malar bone are reported, in one of which the condition was associated with unilateral tumors of the tongue and of the cheek. In both instances the ear canals were narrowed, but in the x-ray films the maxillary sinus seemed to be dilated rather than obliterated.

Fibrous dysplasia of the facial bones is not strictly monostotic, but it may be localized and unilateral.

The microscopic findings differ regarding the distribution of connective tissue, on the one hand, and bone trabeculae, on the other. At present it cannot be stated whether or not the difference indicates variants of fibrous dysplasia.

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12. Woodruff, G. H.: Ossifying Fibroma of the Ethmoid Cells and the Frontal Sinus: Report of Case, Ann. Otol. Rhin. & Laryng. **54**:582, 1945.

13. Fry, W. E., and DeLong, P.: Psammoma of the Orbit, Am. J. Ophth. **24**:664, 1941.

14. Lichtenstein, L.: Polyostotic Fibrous Dysplasia, Arch. Surg. **36**:874 (May) 1938.

15. Lichtenstein, L., and Jaffe, H. L.: Fibrous Dysplasia of Bone: Condition Affecting One, Several or Many Bones, Graver Cases of Which May Present Abnormal Pigmentation of Skin, Abnormal Sexual Development, Hyperthyroidism or Still Other Extraskeletal Abnormalities, Arch. Path. **33**:777 (June) 1942.

16. Schlumberger, H. G.: Fibrous Dysplasia (Ossifying Fibroma) of the Maxilla and Mandible, Am. J. Orthodontics (Oral Surg. Sect.) **32**:579, 1946.

17. Brunner, H., and Grabscheid, E.: Zur Kenntnis der Ostitis deformans (Paget) der Schädelbasis; die vordere Schädelgrube mit besonderer Berücksichtigung der Nebenhöhlen der Nase, Virchows Arch. path. Anat. **301**:237, 1938.

POLIOMYELITIS AND THE LARYNGOLOGIST

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ONLY IN the past two or three years has the laryngologist been considered an essential member of a consultant staff in the treatment of poliomyelitis. Still, in some quarters his position today is challenged, in spite of the increasing evidence in the literature that he offers a therapy that saves lives. His knowledge of respiratory problems seen often from foreign bodies, laryngeal cancer, tracheobronchitis, postoperative atelectasis, and laryngeal edema can be applied directly to the respiratory problems in acute poliomyelitis. As a member of the poliomyelitis team, the laryngologist should observe the patient early, be able to recognize any impending respiratory difficulty, and act accordingly. He should not be considered simply a technician to be called to act on the decision of someone else.

HISTORICAL BACKGROUND

Apparently not until 1932 was there any report that patients with respiratory problems in poliomyelitis could be benefited by treatment. In that year, Wilson,[1] of the Department of Pediatrics, Harvard Medical School, recognized the relief afforded by direct removal of secretions from the respiratory tract. He considered tracheotomy a help in selected cases, although a radical procedure. More experience convinced him, according to reports in 1941 [2] and 1943 [3], that occasionally tracheotomy was lifesaving. In 1936, Davison [4] believed that tracheotomy might help in bulbar poliomyelitis.

More thought and study were given to the problem after the publication of a short article in 1943 by T. C. Galloway [5] a laryngologist of Northwestern University. He recorded his important observation that tracheotomy had saved two patients who otherwise would have succumbed to the disease.

Throughout the country, an increase of the disease in epidemic form in succeeding years gave physicians more opportunity to recognize the respiratory obstruction

Read before the annual meeting of the Pacific Coast Oto-Ophthalmological Society, Victoria, B. C., May 27-31, 1951.

1. Wilson, J. L.: Acute Anterior Poliomyelitis, New England J. Med. **206**:687-893 (Dec.) 1932.

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early, when formerly it was frequently overlooked. The lifesaving effects of tracheotomy were heard of in 1947 from Minneapolis. Priest, Boies, and Goltz [6] reported on the 1946 Minneapolis epidemic of 1830 cases. Approximately 400 patients were given the diagnosis of bulbar poliomyelitis. Of the 75 patients on whom tracheotomy was performed, 29 survived and 19 of these left little doubt that tracheotomy had directly saved their lives. In 1949, Priest [7] appeared before this group to recount his experiences in the 1946 epidemic.

Further studies were reported by Galloway [8] in 1949; he had had 15 consecutive recoveries from bulbar poliomyelitis with six tracheotomies in 127 total cases within three years. The next year, however, he had five deaths despite tracheotomies.

The largest series of tracheotomized patients was reported from the Pacific Coast as a result of the severe 1948 epidemic. Miller and Buck [9] discussed the results of 388 cases of bulbar poliomyelitis with 198 tracheotomies and 101 deaths (28.8%). They consider this mortality rate gratifyingly small. During the years 1946-1949, more than 6,000 patients with poliomyelitis were treated at Los Angeles County General Hospital, with the performance of 357 tracheotomies.[10]

This paper reports the observations of my colleagues and me on 101 patients with acute poliomyelitis admitted to the Fresno County General Hospital, Fresno, Calif., in 1950. In 27 cases diagnoses of bulbar and bulbar-spinal poliomyelitis were made. Tracheotomies were performed on 15 patients.

The problem during the year was one of education. The medical staff, nurses and attendants had to be taught to observe early in patients admitted for poliomyelitis any symptoms indicating a respiratory problem. Although we listed clean-cut indications for tracheotomy, their applications at times were difficult. Early tracheotomy was advised.

Some patients progressed extremely rapidly; others, with marked bulbar signs that seemed to be progressing suddenly recovered. Yet, under close observation, the symptom pattern of respiratory difficulty, aside from the rate of progression, developed similarly in most cases. What was needed more than anything else was careful observation by someone trained to note impending respiratory distress.

Understandably, the attending pediatrician or internist was reluctant to approve performance of what appeared to be a prophylactic tracheotomy, to some physicians a formidable procedure.

There were six deaths among the tracheotomized patients. Our feeling was that the deaths were caused by respiratory complications rather than by a spread of the virus to the bulbar vital centers. Lung involvement, consisting of pneumonia, atelectasis, and pulmonary edema, was found at necropsy in every case. Hemorrhages in the brain which were seen were explained by the asphyxia, not necessarily by action of the virus. One cannot state for certain that all the deaths were due to

6. Priest, R. E.; Boies, L. R., and Goltz, N. F.: Tracheotomy in Bulbar Poliomyelitis, Ann. Otol. Rhino. & Laryng. **56**:250-263 (June) 1947.

7. Priest, R. E.: Tracheotomy in Bulbar Poliomyelitis, Tr. Pacific Coast Otol.-Ophth. Soc. **30**:144-156, 1949.

8. Galloway, T. C., and Seifert, M. H.: Bulbar Poliomyelitis, J. A. M. A. **141**:1 (Sept. 3) 1949.

9. Miller, A. H., and Buck, L. S.: Tracheotomy in Bulbar Poliomyelitis, California Med. **72**:34-36 (Jan.) 1950.

10. West, H. E.: Treatment of Poliomyelitis with Involvement of the Respiratory System, California Med. **73**:397 (Nov.) 1950.

asphyxia, as the findings are identical in encephalitis. Many times the tracheotomies were delayed because of late admissions. Lung complications had already supervened. However, the pulmonary diseases in some of the cases were reversible with therapy.

PATHOLOGICAL PHYSIOLOGY

Respiratory obstruction in poliomyelitis may be caused by several mechanisms. Secretion obstruction is predominant. Central stimulation of the salivatory nuclei in the medullary bulb due to anoxia or inflammation has been suggested as an important cause of the hypersecretion. Peripheral factors must also play a part. Normally at least a quart (950 cc.) of fluid is daily produced in the respiratory tree. Loss of the cough reflex and loss of the swallowing mechanism cause accumulation of the fluid which normally is expelled. Also, edema of the lung and tracheobronchial inflammation produce increased fluid.

Loss of the cough reflex follows from the peripheral paralysis of the intercostal and abdominal muscles and the diaphragm. The patient is unable to inspire air and hence to increase the intrathoracic pressure as he must before he can cough. If a patient can cough effectively, he does not have complete paralysis of the respiratory muscles.

The coordination of the swallowing mechanism is complicated and easily disturbed centrally. Peripheral nerve paralysis of the 5th, 9th, 10th, 11th, and 12th cranial nerves leads to loss of function of the pharynx, tongue, floor of the mouth, and larynx, with consequent interference with swallowing. Normally, with swallowing the larynx moves upward, closing the glottis. With the laryngeal sphincter function destroyed, food and secretions pour over into the airway. Coughing also is dependent upon proper laryngeal function.

Flaccid vocal cords from vagal paralysis contribute another form of breathing obstruction by a ball-valve-like action. With inspiration the cords are drawn below, closing the airway. With expiration the cords are forced upward.

A patient placed in a respirator, being unable to cough, has secretions accumulate in his respiratory tree. Normally, breathing ceases at the end of expiration during swallowing. The paralyzed person in the respirator cannot stop breathing to swallow. As a result, the incoming air carries liquids and foods into the trachea with each attempt at swallowing. Even with no bulbar symptoms, the patient with complete paralysis of the respiratory muscles may need a tracheotomy. Secretions also accumulate from inflammation and pulmonary edema. In other words, tracheotomy is not necessarily confined to patients with bulbar poliomyelitis.

These respiratory obstructions in acute poliomyelitis, of course, may swiftly lead to a fatal termination in the space of a few hours. An apparently compensated state in which the patient appears to be resting comfortably may suddenly, in minutes, progress to an asphyctic crisis.

In a simple obstruction without as yet any secondary lung complications physiologically, there occurs first a reduction in the ventilation capacity of the lungs. Dyspnea on exertion is the only symptom. The hypoventilation leads to an accumulation of carbon dioxide (hypercapnia). Further progression induces a decrease in the oxygen saturation of the arterial blood (anoxemia).

A fall in the blood pH (acidemia) because of excess carbonic acid brings about a respiratory acidosis. If the obstruction persists, the tissues starved for oxygen

produce excess lactic acid. Its accumulation in the blood is termed a metabolic acidosis. All these changes contribute to the asphyxia that may not be reversible if intervention is late.

A hastened course of events ensues from lung complications secondary to the obstruction. Infection of the lungs, atelectasis from blockage of bronchi and bronchioles, and pulmonary edema further increase the accumulation of carbon dioxide and further lower the arterial oxygen saturation, with severer acidosis and more rapid asphyxia.

The blood changes early actually increase the stimuli to the breathing apparatus. The observer may be lulled into believing that no respiratory difficulty is present. Inhibition of breathing may arise only when the blood changes can no longer be compensated. An abrupt and fatal outcome is witnessed.

SYMPTOMATOLOGY

Familiarity with the symptoms associated with inadequate respiratory exchange may prevent many deaths. Headache, increased mental activity, agitation, sweating on the forehead, dyspnea, uncooperativeness, anxiety, and restlessness are noted early. Euphoria at times also is seen. Later with progression of events, confusion disorientation, and irrationality are observed, leading to convulsions, unresponsiveness, lethargy, and coma.

When we see a patient becoming antagonistic toward our efforts to help him and showing combativeness toward the attendants, we immediately suspect interference with respiration. Sedative drugs, especially opiates, are contraindicated, since the patient is struggling for all the oxygen he can get. Increase in the pulse rate and a very high blood pressure frequently accompany this condition. Cyanosis is not a signal to be waited for to perform surgery. When it occurs, the prognosis is much more uncertain.

One little boy whom I have observed, when first seen was leaping up in bed, flinging his arms about with a wild, starry-eyed appearance. A large amount of froth welled up from his mouth. The child was alert but could not speak. He appeared enraged at his inability to remove the mucus. Any fluids given him were regurgitated through the nose.

Too many times symptoms are blamed upon the encephalitis accompanying the disease. Difficulty arises because the symptoms are similar in respiratory obstruction and in encephalitis. Only when we are certain that the airway is clear and the pulmonary acidosis corrected can we ascribe symptoms to the encephalitis.

Also, the anoxia is not the important cause for the symptoms. Studies by the United States Air Force [11] revealed that decreasing the oxygen intake may produce unconsciousness without distress or premonition of danger. However, breathing 10% carbon dioxide without any oxygen lack induces extreme distress, apprehension, and fear of impending death. In personnel given longer exposures and higher concentrations of carbon dioxide anesthesia, respiratory depression, convulsions, narcosis, and circulatory collapse developed. In asphyxia, the anoxia or deficiency of oxygen compared with the respiratory acidosis represents only part of the picture.

11. Gray, J. S.: Physiology of Respiratory Obstruction, Ann. Otol. Rhin. & Laryng. **59**:72-77 (March) 1950.

TREATMENT

Early intervention in the relief of respiratory obstruction is vigorously advocated. Because of the abrupt and unexpected change possible in the patient's condition, conservative management of respiratory obstruction is contraindicated.

Tracheotomy is immediately considered when doubt exists that the airway can be constantly kept clear. Postural drainage with the patient in the Trendelenburg position and aspiration through the nose and mouth are not sufficient when the patient has any symptoms, however slight, suggesting respiratory difficulty. The many deaths before tracheotomy attest to the failure of postural drainage and aspiration. Oxygen, although important, will not correct the increased carbon dioxide concentration and acidosis.

Inability to swallow or cough denotes an existing or impending secretional obstruction and calls for surgical action. Spasm or paralysis of the vocal cords is another indication. With pulmonary edema or atelectasis already existent, we can never hope to relieve the patient without tracheotomy. Every patient who is in a respirator and has bulbar symptoms requires tracheotomy because of the danger of sucking secretions down into the lungs. Previously, respirator therapy was contraindicated in bulbar poliomyelitis. An almost certain death was expected. Now, with tracheotomy, the respirator finds its greatest usefulness in bulbar poliomyelitis.

One of our early patients, an 8-year-old boy, seemed comfortable with aspiration and postural drainage, although he was unable to swallow. He was becoming increasingly restless and suddenly manifested generalized convulsions, cyanosis, coma, and stopped breathing. A resident hastily passed a Flagg laryngoscope and with it separated the spastic vocal cords. The patient began to breathe but continued to have convulsions. Gradually his color improved, but he remained comatose. A tracheotomy was performed about 10 minutes later, with considerable aspiration of secretions. He was placed in a respirator. Within 30 minutes he was conscious, with good pulse and color. With watchfulness and application of the indications for tracheotomy, such an asphyctic crisis would not have occurred.

Advantages of tracheotomy, besides the assurance of a clear airway by direct suction, include the ease with which bronchoscopy can be done as frequently as necessary through the wound for removal of secretions from the lower bronchial tree. Every respirator patient who had undergone tracheotomy received oxygen through the tube by means of an adapter and the Bennett flow-sensitive intermittent positive pressure unit. This apparatus, we felt, is more advantageous than periodic bronchoscopy in keeping the lungs expanded. Atelectasis and pulmonary edema occur much less often with the use of a positive-pressure unit.

Tracheotomy is always done at the level of the second tracheal ring. If lower, the respirator collar, especially in adults, falls over the tube and interferes with subsequent nursing care. Rarely is it necessary to sever the thyroid isthmus, which lies over the second ring. By a transverse incision over the thyroid fascia and its separation from the isthmus, the gland can easily be retracted from the field. In this fashion, considerable bleeding is avoided. Fascial dissection away from the midline is not done. All tracheotomies, of course, were done with local or no anesthesia, depending upon the condition of the patient.

No sutures are placed. Leaving the wound open reduces the danger of infection and prevents air from entering the tissue spaces, with subsequent mediastinal emphysema, not uncommon in infants.

In only two cases did we perform the tracheotomy over a bronchoscope. In an apprehensive, struggling patient with no laryngeal obstruction, no need exists, even in infants, to pass a bronchoscope previous to surgery. Our positive-pressure unit with face mask is helpful in complete respiratory paralysis. We avoid doing the tracheotomy under trying circumstances with the patient in a closed respirator. The unit itself maintains adequate respiration.

No postoperative complications in any of the cases followed the surgical procedure. Tracheotomy is a safe procedure, much safer than delay until conditions become irreversible. After-care by trained personnel who have been instructed to apply suction gently and deeply at frequent intervals helps immensely to tide the patient over his acute symptoms. Cyanosis developing in spite of a clear airway suggests atelectasis or pulmonary edema. Frequent roentgenograms are taken, followed by bronchoscopy if necessary. Antibiotics given routinely effectively prevent secondary lung infection. Frequent turning of the patient is done to insure that all portions of the lung are aerated. Plasma is given intravenously because of disturbance of the albumin-globulin ratio. All feedings, for the first two days or so, are parenteral to prevent fluids and foods from entering the airway. Also, abdominal distention may result from early oral feeding. Wangensteen suction relieves abdominal distention and vomiting, preventing fluids and foods from entering the mouth.

Decannulation is not done until it is certain that all the bulbar symptoms have disappeared and that the cough reflex has returned. At least three weeks is required before the tube is removed.

SUMMARY

The place of the laryngologist in the poliomyelitis team is becoming assured through the increasing evidence that he offers a therapy that saves lives. This paper reports observations upon 101 patients with acute poliomyelitis admitted to the General Hospital of Fresno County, Fresno, Calif., during 1950. Fifteen tracheotomies were performed, with six deaths. Education of the staff to recognize respiratory distress helped to reduce the mortality.

Tracheotomy is a lifesaving procedure to relieve respiratory obstruction caused by hypersecretion, by loss of the laryngeal sphincter action with spillage of food, fluids, and vomitus into the airway, and by flaccid vocal cords. Respirator patients present a problem because of the loss of the cough reflex and may require tracheotomy.

Respiratory acidosis from accumulation of carbon dioxide and metabolic acidosis from oxygen-starved tissues produces a progressive symptom pattern leading to coma and death unless intervention to relieve the breathing obstruction is done.

A victim of poliomyelitis who is restless and uncooperative may have interference with his airway and needs immediate treatment. Tracheotomy is a safe procedure, to be performed early, and is more effective than postural drainage and aspiration alone.

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HEARING LOSS IN CHILDREN WITH CLEFT PALATES

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THE RELUCTANCE of most otolaryngologists to perform tonsillectomies and adenoidectomies on children with cleft palates has been one of the factors diverting the interest and attention of these specialists from a problem which vitally needs the benefits of their skills. The object of this investigation is chiefly to evaluate the effectiveness of present-day management of tonsils and adenoids in patients with cleft palates. The study was motivated by a request from Dr. Frank P. Bakes, head of the speech clinic at the University of Pennsylvania, to determine whether or not hearing loss was an important factor in speech development and speech correction in children with cleft palates. This paper is a preliminary report until more cases have been studied and will concern itself not with surgical technique, which has been so well advanced by Ivy,[1] Brophy,[2] Beatty,[3] Davis,[4] Royster,[5] and others, but with surgical judgment.

The generally accepted concepts governing the management of tonsils and adenoids in children born with cleft palate have been clearly described by Beatty.[3] Complications in the ear and obstructed respiration due to adenoid hypertrophy are the chief indications for surgery. The general consensus, however, is to refrain from doing a tonsillectomy and adenoidectomy on children with cleft palates until they reach the age of 6 or 7 years whenever possible. "If tonsillectomy is done," Beatty states, "by any of the accepted methods, natural contractions will, to varying extents, impair the speech and function of the velum in deglutition." He, therefore, has devised a special technique which may, at times, improve the speech and not affect the normal functioning of the palate.

The abnormal condition to which the Eustachian tube is exposed in cleft palate makes it a constant object of infection from food, trauma, and inadequate aeration. Beatty feels that infants with cleft palate possess some natural protective factor to prevent infections and suggests that the factor may be a reflex closure mechanism along with the bacteriostatic action of the mucosa in well-cared for infants. In his experience, the majority of well-fed and well-cared for infants do not have ear

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1. Dr. Robert Ivy is Chief of the Cleft Palate Division, Department of Health, Harrisburg, Pa.

2. Brophy, T. W.: Cleft Lip and Palate, Philadelphia, The Blakiston Company, 1923.

3. Beatty, H. G.: Cleft Palate, Ann. Otol. Rhin. & Laryng. **55**:572 (Sept.) 1946.

4. Davis, W. B.: Harelip and Cleft Palate, Ann. Surg. **81**:536 (Jan.) 1925.

5. Dr. H. P. Royster: Personal communication to the authors.

infections prior to the age for surgical closure of the palate. Day [6] has repeatedly emphasized that one of the commonest causes of otitis media in infants is feeding them while they are in the supine position. The constant exposure of the Eustachian tube in cleft palate certainly must make this cause a most significant one very early in infancy. In my experience, examination of the ears of very young children with cleft palate reveals a high incidence of pathological changes, despite the absence of subjective symptoms of otitis media.

Thirty consecutive patients with cleft palate were carefully studied in this investigation. Twenty-two patients were from the State Cleft Palate Clinic of

Findings in Children Ranging in Age from Five to Sixteen Years

Case No.	Age, Yr.	Otitis	Deafness	Membrani Tympani	T. & A.*	Palate	Tonsils	Adenoids	Cervical Nodes
1	8	Yes	Yes	Path.	No	Not repaired	Hypert.	Hypert.	Sm. bilat.
2	14	Yes	Yes	Path.	3 yr.	Repaired	None	Scant	Sm. bilat.
3	4	Yes	?	Path.	No	Repaired	Hypert.	Hypert.	Lg. bilat.
4	9	No	Yes	Path.	No	Repaired	Hypert.	Hypert.	Sm. bilat.
5	7	Yes	Yes	Path.	3 yr.	Repaired	None	Moderate	Sm. bilat.
6	8	Yes	Yes	Path.	No	Repaired	Hypert.	Moderate	Sm. bilat.
7	9	No	No	Path.	No	Prosthesis	Small	Moderate	Lg. bilat.
8	9	Yes	Yes	Path.	No	Repaired	Small	Moderate	Sm. on rt.
9	11	No	Yes	Path.	No	Repaired	Hypert.	Hypert.	Lg. bilat.
10	12	Yes	Yes	Path.	9 yr.	Repaired	None	Scant	None
11	11	No	Yes	Path.	No	Repaired	Hypert.	Scant	None
12	4	Yes	?	Path.	No	Repaired	Hypert.	Hypert.	Lg. bilat.
13	7	No	Yes	Path.	No	Repaired	Small	Moderate	None
14	9	No	No	Path.	9 yr.	Repaired	None	None	None
15	15	No	Yes	Path.	No	Repaired	Hypert.	Moderate	None
16	12	No	Yes	Minimal	No	Prosthesis	Hypert.	Hypert.	Bilat.
17	6	Yes	Yes	Path.	No	Repaired	Hypert.	Hypert.	Bilat.
18	7	Yes	Yes	Path.	No	Repaired	Hypert.	Hypert.	Bilat.
19	8	Yes	Yes	Path.	No	Repaired	Hypert.	Hypert.	Sm. on lt.
20	7	Yes	Yes	Path.	No	Repaired	Hypert.	Hypert.	Bilat.
21	16	Yes	Yes	Path.	No	Repaired	Hypert.	Hypert.	None
22	7	Yes	Yes	Path.	No	Not repaired	Hypert.	Hypert.	Bilat.
23	6	Yes	Yes	Path.	No	Repaired	Small	Hypert.	Bilat.
24	8	No	Yes	Path.	3 yr.	Repaired	None	Hypert.	Bilat.
25	5	No	Yes	Path.	No	Repaired	Small	Hypert.	Bilat.
26	9	Yes	Yes	Path.	7 yr.	Repaired	None	Moderate	Bilat.
27	6	Yes	Yes	Path.	No	Repaired	Hypert.	Hypert.	Bilat.
28	16	Yes	Yes	Path.	No	Repaired	Hypert.	Moderate	Sm. bilat.
29	9	Yes	Yes	Path.	No	Repaired	Hypert.	Moderate	Bilat.
30	13	Yes	No	Path.	6 yr.	Repaired	None	Scant	Sm. on lt.

* Tonsillectomy and adenoidectomy.

the Children's Hospital, Philadelphia, and eight were private patients. There is always a tendency to assume that clinic patients do not fall into the category described by Beatty as "well fed and well cared for," but my experience does not justify this. All these patients received complete examinations, including otological, speech, dental, psychological, and physical measurements for growth research studies by Dr. Wilton Krogman. All the patients were given at least two audiometric tests, each one by a different audiologist, in a soundproofed room, using a calibrated audiometer. Whenever there was inconsistency, further hearing studies were done until an accurate threshold was established, if possible. The

6. Day, K. M.: Management of Deafness: Wherry Memorial Lecture, Tr. Am. Acad. Ophth. **55**:22, 1950.

difficulty in doing accurate audiometric tests on 5-year-old children is recognized, but in all children in this consecutive series reasonably good thresholds were obtained unless stated otherwise.

The table shows the findings. The ages ranged from almost 5 to 16 years. Column 3 states whether or not a history of otitis media or earache was elicited from the mother. Column 4, marked "Deafness," designates the audiometric results. "Yes" means that a hearing loss of at least 15 db. was present in at least two frequencies. In column 5 is stated the appearance of the membrana tympani. "Path." designates a pathological change in the appearance of the drum head and middle ear. The pathological change had to be clearly evident to fit into this classification. Criteria were such changes as retraction, perforation, scars, marked thickening, and opacity. If a semblance of the cone of light was present, the ear was not included in the pathological group but marked "minimal." Column 6 states whether or not the patient had experienced surgery for a tonsillectomy and adenoidectomy and, if so, at what age. Column 7 states whether or not palatal surgery had been performed. The next three columns describe the present condition of the tonsils, adenoids, and cervical nodes. Nasoscopic examinations were done in only a few patients, since in most of them described as having adenoid hypertrophy the lymphoid mass could be seen through a cleft in the palate or protruding well below the repaired and retracted palate.

COMMENT

Ten of the 30 patients gave no history of symptoms referable to the ear, despite the fact that all showed pathological change in the middle ear. This was not unexpected, since examination of at least 20 other infants with cleft palate, not included in this study, revealed that many of them had signs of middle ear pathological change. It is my impresssion that this type of change is due not so much to the presence of infection in the ear as it is to disparity of pressures between the outside and the middle ear. Many of the children with cleft palate have considerable difficulty aerating their middle ears in infancy and this, along with the abnormal exposure of the Eustachian orifice, tends to facilitate early insidious hearing loss without subjective symptoms.

Only three of the entire series of patients showed normal hearing on the audiogram, but all three children showed middle ear pathological changes. It is well known that the pathological change visible is no indication of the degree of hearing impairment and vice versa. With the exception of patient 16, who showed stria in both drum membranes but had a dull cone of light, every ear examined in this series was definitely pathological. Patients 3 and 12, for whom accurate thresholds were not obtainable, revealed pathological change of the middle ear which was so severe that the presence of hearing loss is presumptive. Both these children had evidence of acute otitis media with bulging and inflamed ear drum membranes during their first examination.

The degree of hearing impairment varied from an average of 45 db. to minimal loss. The predominant loss in hearing acuity was in the low frequencies. Patient 15 exhibited an interesting finding of possible bilateral congenital nerve deafness along with the cleft palate. No definitive etiology was obtainable. In most of the patients the hearing loss was bilateral and often followed similar patterns in both

ears. In patient 21, the loss was principally in the higher frequencies of 4000 and 8000 cps with minimal loss in the low frequencies. The ear drum membranes in this patient were extremely distorted with scars and markedly retracted. The hearing loss in all the patients, except patients 15 and 21, was characteristically conductive.

Only seven of the patients had had their tonsils and adenoids removed. One of these, patient 9 had no hearing loss, but definite pathological change in both ears was present. His tonsils were very recently removed. With few exceptions, the tonsillar and adenoid tissues were hypertrophied in all the children in whom no tonsillectomy and adenoidectomy had been done. Most of them had palpable cervical nodes.

The startling facts that over 90% of this series of children with cleft palates revealed hearing loss and 100% showed pathological changes in both middle ears make it necessary to question the effectiveness of present management of tonsils and adenoids in such situations. Deafness and ear infections must be given greater importance in determination of the over-all management of these children.

Since Beatty's principal emphasis has been on tonsillectomy and its effect on speech and deglutition, until further evidence is made available which refutes his concepts it is probably wise to delay tonsillectomy until the age of 6 years, whenever possible. With respect to the adenoid tissue, however, a completely new approach is suggested. Dr. George M. Coates, who gave invaluable advice during this investigation, on reviewing the results suggested the importance of preventing ear complications and nasal obstruction due to hypertrophied adenoids in children with cleft palates by a carefully performed adenoidectomy as early in infancy as possible. Discussion with Dr. Bakes indicates that this procedure would probably have no harmful effect on speech. Performance of an excellent adenoidectomy on a child with a cleft palate is much easier and safer than waiting until the palate is repaired and, of necessity, operating without visualization of the nasopharynx owing to the inability to retract the repaired palate.

CONCLUSIONS

To prevent some middle ear complications and nasal obstruction due to hypertrophied adenoid tissues in children with cleft palates, adenoidectomy carefully performed in infancy, preferably at the time of the first operation for surgical closure of the palate has been suggested. Removal of the tonsils is probably not advisable at the same time but should be delayed until the age of 6 years, if possible.

SUMMARY

A preliminary study of 30 children with cleft palates reveals pathological findings in the middle ears and conductive hearing impairment in almost all of these children. Since hypertrophied adenoids may sometimes contribute to otitic complications, the advisability of doing careful adenoidectomy in infancy prior to palatal closure is being considered.

Case Reports

LUPUS ERYTHEMATOSUS DISSEMINATUS

Report of a Case with Five-Year Arrest Following Ethmoidectomy

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A CASE was observed in which lupus erythematosus disseminatus existed concurrently with a chronic ethmoid infection. With relief of the ethmoiditis through surgery, the lupus symptoms disappeared and the patient was able to resume an active life.

REPORT OF A CASE

On April 16, 1945, Miss M. R. sought my advice. She had been suffering since 1941 from attacks of erythema on exposed skin surfaces, swollen painful joints, headaches, and malaise, associated with reduction in both white and red blood cell counts. During the winter of 1940 and 1941 in New York she was asked by friends whether she was sunburned. She had felt exhausted during this period; the cause was unknown. In May, 1941, she had an acute attack of moderate severity, with skin lesions, joint swelling, and fever, following prolonged exposure to sunlight. In January, 1942, she went to the Mayo Clinic (service of Dr. B. R. Kierland) and was hospitalized. A diagnosis of subacute disseminated lupus erythematosus was made. The results of blood studies were relatively normal, showing a white cell count of 6,500 which was reduced to 2,500 after bismuth injections. The sedimentation rate varied between 30 and 58 mm. per hour. The patient was advised to return home and remain in a darkened room, suggestions which she followed for four months. In June, 1942, she returned to the Mayo Clinic and was told never to be exposed to sunlight again. She returned home, and up to the time that my observation began she had spent three-fourths of her time in bed with malaise, frequent fever, headaches, and swollen joints.

The course of the illness is outlined in the following report of the hospital admissions in the service of Dr. J. O. Ritchey and Dr. James Browning, internists, and Dr. John Dalton, dermatologist, at the Methodist Hospital, Indianapolis.

1. The patient was admitted on Sept. 14, 1942, acutely ill, with a temperature of 103.8, skin lesions, headache, and painful joints. At this time the laboratory findings were as follows:

Sedimentation Rate: Vertical curve:
Time 20 min.
Index 28 min.

The red blood cells numbered 4,200,000; the white blood cells, 1,600; the hemoglobin content was 13 gm. Three blood transfusions were given on successive days as well as sulfadiazine medication, 1 gm. every four hours. After this therapy the red blood cells numbered 4,320,000 and the white blood cells, 3,050. Urinalysis showed an occasional trace of albumin and blood. Bone marrow observation obtained through a sternal puncture showed the marrow to be hyperplastic, with the following arrangement as to differential: Basophilic polymorphonuclear leucocytes; segmental forms; metamyelocyte band forms, 3; myelocytes C, young band forms, 59; myelocytes B, young band forms, 11; myelocytes A, young band forms, 5.

The red blood cell count taken on Sept. 18, 1942 showed 3,400,000 red cells; 3+ reticulated forms; 3+ neuroblasts; 3+ erythroblasts; 4+ megaloblasts; anisocytosis, poikilocytosis, and achronia; and 4,100 white cells.

2. On Oct. 14, 1942, the patient was again admitted to the Methodist Hospital for blood study and two transfusions. At that time, before the transfusions, the red blood cells numbered 3,030,000; the white blood cells, 2,700; the hemoglobin content was 11.5 gm. Polymorphonuclear leucocytes comprised 80%. Skin biopsy at this time showed myxedematous tissue.

3. On Dec. 6, 1942, the patient was again admitted for blood count and blood transfusions and was discharged on Dec. 11, 1942.

4. On Dec. 28, 1942, the patient was admitted again for similar procedure and discharged on Jan. 1, 1943.

5. On March 3, 1943, the patient was again admitted for a four-day period of observation and blood transfusions.

6. On Jan. 27, 1945, the patient was admitted to the hospital again for three blood transfusions covering several days' time. She had been observed outside the hospital and the blood levels had been sustained in a fairly satisfactory degree until this date.

7. On March 12, 1945, because of her general condition, it was thought wise to admit the patient again for more blood transfusions.

8. On May 3, 1945, the patient was admitted with an exacerbation of the symptoms mentioned at her first hospital admission. A throat culture showed Gram-positive cocci in chains and groups, with Micrococcus pyrogenes var. aureus (Staphylococcus aureus). The patient was given penicillin therapy, after which her general symptoms improved promptly.

The patient sought my advice because she had noted that each exacerbation was preceded and accompanied by symptoms of active inflammation of the intranasal structures. These were obstruction to breathing and headache of ophthalmic distribution, both worse on the right side.

My diagnosis was a high septal deviation to the right with blockade and infection of the ethmoid sinuses, more active on the right side. In our observation over several months, her headache was relieved after shrinking of the sinuses each time. Both the patient and I became convinced that chronologically the activity of her systemic disease in each instance was associated with activity in a chronic ethmoiditis. Furthermore, the use of penicillin in each case seemed to control the attack, which suggested that the infection was an important link in the chain of her lupus syndrome. A cause-and-effect relationship between her ethmoid infection and the exacerbation of her lupus was very strongly suggested for the following reasons:

1. The infection seemed to be fundamental in her symptom complex (improved by penicillin therapy). 2. A chronic infection was present in her ethmoid sinuses. 3. Her intranasal infection became active just before a systemic attack, her headache was relieved on shrinking of the ethmoid sinuses and she felt decreased intranasal congestion. 4. Systemic damage from blockade of an infected ethmoid is often very great if the infective agent has properties to which the patient is sensitive. I am convinced of this in my observation through the years of over 1,000 cases in which ethmoidectomy was performed.

The patient urged surgical correction, as nothing short of this would eradicate the infection. I was discouraged from this course by her medical advisor, who felt that patients with lupus erythematosus were poor surgical risks. The patient, however, did not care to live in her present condition and was willing to assume the risk. I also felt that with the support of penicillin therapy the outcome would be favorable.

On July 9, 1945, I performed a submucous resection and right ethmoid exenteration. The postoperative course with the above medication was smooth, and the patient's recovery uneventful. The right-sided headache ceased, and general improvement ensued. On Oct. 30, 1947, an ethmoidectomy was performed on the left side because of an occasional headache behind the left eye, due to a moderate amount of infection in the left ethmoid and sphenoid sinuses. Bilateral maxillary sinus windows were also made.

As of March, 1951, nearly six years since the operation, the patient has been clinically free of all signs of lupus erythematosus. She has been in bed with an acute cold (accompanied each time by slight joint tenderness), but no skin changes, only a few days since her operation. Her headaches are absent. She lives a busy, useful life operating an advertising business in a downtown office eight hours a day.

CONCLUSION

This is simply a case report and presented for whatever it may be worth. I do not conclude that lupus erythematosus is caused by infection. It does seem, however, that in this case certain changes were produced in the patient's economy by the removal of the toxic load of her infection and that these changes were fundamental in the arrest of her disease. Lupus erythematosus as one of the so-called collagen diseases is thought to be associated with a response of an allergic nature. It is conceivable that the allergen might be a bacterial protein and that when the allergen is no longer present the chain of events which activates lupus no longer operates.

I regret that my experience with lupus erythematosus is limited to only three cases. One is reported above. A second showed no intranasal pathology. In the third case the patient refused operation and has disappeared from observation.

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Clinical Notes; New Instruments, and Techniques

SULFAMYLON® IN THE TREATMENT OF EXTERNAL OTITIS

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Sulfamylon® is rapidly gaining popularity in the local treatment of ear, nose and throat infections as a very effective bactericidal and bacteriostatic agent. It is a sulfanilamide derivative with a chemical formula of $SO_2NH_2C_6H_4CH_2NH_2HCL$; its chemical name is para-(aminomethyl)-benzene sulfonamide hydrochloride. It is a white crystalline material, soluble in water, and weakly acid in reaction. Although it is a sulfonamide derivative, research chemists have altered the sulfonamide structural formula to produce sulfamylon®; unlike the original drug, it is nontoxic and is active in the presence of paraaminobenzoic acid, blood, tissue juices, and pus.

The in vitro experimental work was done to a large extent by Brewer,[1] who compared concentrations of sulfamylon® and sodium sulfadiazine and sulfathiazole. In this work he found that sulfamylon® had a much greater bactericidal activity than either of the other two solutions. The solution was also found to be quite nontoxic whether administered orally, subcutaneously, or intravenously. In their research Hamre and her associates [2] demonstrated that it had an extremely low lethal dose when taken internally either by injection or by oral administration. Hamre [2] also found that solutions up to 2% had no effect on the conjunctival or bladder mucosa.

Clinically, several papers have been published on the reactions and effects of sulfamylon® on other parts of the body. Howes [3] and his associates reported an investigation of the toxicity to tissues and its speed of action on wound infections. He found that it was active in the presence of pus and blood and was not affected by a change in the acidity of the environment. He maintains, however, that the treatment of open wounds containing slough and sequestrum was unsuccessful. Brewer [1] also used this substance locally as a prophylaxis against infection and in the treatment of infected wounds and came to the conclusion that it was a valuable aid in this type of therapy. Clark,[4] too, used it in a 1% solution as a prophylaxis against the Pseudomonas aeruginosa (Bacillus pyocyaneus) in preoperative ophthalmic cases. So far as the therapy of ear, nose, and throat infections is concerned, Fox [5] in 1947 carried on an investigation of the use of sulfamylon® in rhinosinusitis. He reported that it was effective against all the organisms present, having its poorest effect on Ps. aeruginosa, and in none of the cases did he observe any constitutional reaction. Diehl and Morris [6] in 1949 reported its use in treatment of ear infections and also found it effective. In our work reported at that time [6] we found that it was comparatively ineffective in acute external otitis cases. This is in direct contradistinction to the conclusions of McLaurin,[7] who found it effective in external otitis. However, in our work [6] we had used this solution in only a few cases. As the following report will indicate, a follow-up of this local treatment of external otitis demonstrates sulfamylon® to be effective.

In the technique of the treatment of external otitis, we followed the procedure of McLaurin.[7] First, the entire ear canal was cleansed as thoroughly as possible; second, the entire ear canal

1. Brewer, J. H.: Clear Liquid Mediums for the "Aerobic" Cultivation of Anaerobes, J. A. M. A. **115**:98 (Aug. 24) 1940.

2. Hamre, D. M.; Walker, H. A., and others: Proc. Soc. Exper. Biol. & Med. **55**:170, 1944.

3. Howes, E. L.: Ann. Surg. **124**:268, 1946.

4. Clark, W. B.: Local Use of "Sulfamylon" (Para-[aminomethyl]-Benzene Sulfonamide Hydrochloride), Arch. Ophth. **38**:682 (Nov.) 1947.

5. Fox, S. L.: Ann. Otol. Rhin. & Laryng. **56**:946, 1947.

6. Diehl, K. L., and Morris, A. G.: Sulfamylon® and Streptomycin in Treatment of Infections of the Ear, a Preliminary Report, Arch. Otolaryng. **50**:700 (Dec.) 1949.

7. McLaurin, J. W.: Laryngoscope **58**:1201, 1948.

was painted with 95% alcohol. In those cases in which irritation produced a severe burning as a result of the application of the alcohol, a stream of air was gently introduced into the canal until the burning subsided. The object of this cleansing and the application of alcohol is the thorough drying of all parts of the ear canal, because it has been shown that a moist surface containing macerated tissue is an excellent culture medium for the growth of bacteria. Third, the canal wall was painted with a solution of 5% sulfamylon,® and then a wick of cotton soaked in it was inserted into the canal. The patient was then given a prescription for a 1% solution. At the end of six hours, the patient removed the wick and instilled the sulfamylon® into the ear canal, approximately 4 drops every three hours. The introduction of the wick into the ear canal had the advantage not only of applying the medication in close contact with the infected surface but also of separating any edematous membrane surfaces from actual physical contact. The patient was then requested to return to the office in 48 hours.

In many cases, the time elapsed was more than 48 hours; consequently, we had of necessity to judge the elapsed time of treatment from the first to the second office visit. Several of the patients had definite improvement before making the second office visit.

EVALUATION OF CASES

A total of 30 cases was evaluated. Inasmuch as we were interested only in the clinical results and the time required for cure, no cultures or other laboratory studies were undertaken. Clinical improvement was judged as 100% when the ear canal was clean, with no injection of the walls or soreness in or around the auricle; abatement of pain and swelling, with some pus, persisting injection of the canal wall, or soreness about the auricle was classed as about 50% improvement. The cases were judged in this manner, rather than by the time of final cure, so that the rapidity of improvement could be generally approximated and the efficiency of the medication could be more clearly seen. The following tabulation demonstrates the number of cases and the average percentage of improvement.

		Number of Cases				
Observation Period, Days	Total	With No Improvement	With 50% Improvement	With 75% Improvement	With 100% Improvement	Per Cent of Total
2	9	..	..	1	8	30
3	9	..	..	2	7	30
4	5	..	..	2	3	17
7	5	..	2	1	2	17
14	1	1	..	..	..	3
No return	1	1	..	..	..	3

In evaluating these cases, we were extremely conservative in our judgment. Many patients could have been classed as cured before their second visit, on the third or fourth day. Several had no symptoms but had minimal injection of the canal wall, and consequently were judged as 75% cured. Nevertheless, it can be seen that 94% of the patients showed definite improvement or cure in less than one week, 77% in less than four days, and 60% in less than three days. These percentages are shown with the full realization that the total number of cases is small. However, the consistent good results of the therapy in this small series, we believe, justify the statement that these results are reasonably accurate.

CONCLUSIONS

Sulfamylon® is an effective local therapeutic agent in external otitis. It should be considered as a valuable adjunct in local treatment of external otitis, either acute or chronic.

277 Alexander St. (7).

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1950

OTITIS MEDIA AND COMPLICATIONS

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THERE were a number of interesting contributions to otolaryngology in 1950, one of which was the symposium on penicillin in severe otolaryngological complications prepared by the Danish, Norwegian, and Swedish Otolaryngological Societies. The material was made up from 421 replies to questionnaires from 50 different departments and clinics. Another interesting symposium was held by the Otological Section of the Royal Society of Medicine, in which the theories of the formation of cholesteatoma were discussed. Different aspects of these papers will be discussed under separate headings. Comments of the reviewer appear in brackets.

DIAGNOSIS

Senturia, Matthews, and Adler [1] studied the secretions in 175 cases of external otitis and otitis media. They found, to their surprise, that in acute or chronic external otitis there were produced enormous numbers of epithelial cells and bacteria but no neutrophiles or lymphocytes. Eosinophiles were also rarely found. They explained this lack of pus cells by assuming that the inflammatory reaction involved the superficial layers only. In a very few cases a few pus cells were seen, and the authors assumed that the superficial layers had been trespassed. There was found no way to differentiate the various categories of external otitis—seborrheic and neurogenic otitis, otomycosis, etc. There were many neutrophiles, lymphocytes, and strands of mucus found in cases of otitis media, and they decided that the examination of the secretions is of importance in making a quick differential diagnosis between otitis media and external otitis.

BACTERIOLOGY

Bjuggren and Tunevall [2] followed 178 cases of acute otitis media for five months with the idea of estimating the importance of the Pfeiffer bacillus (Hemophilus influenzae). In all the cases the patients were children under 16 years of age. They found that the infections due to Pfeiffer bacilli were very apt to recur. The percentage of recurrence in this group was 68, as compared with 18.5 in the entire group. They made the observation that, since the entire group was treated with penicillin or one of the sulfonamides, it is possible that the high incidence of Pfeiffer bacilli was due to the fact that the more penicillin-sensitive organisms were destroyed early in the treatment. The great majority of the children with Pfeiffer otitis were under the age of 4 years. [A carefully done investigation.]

1. Senturia, B. H.; Matthews, J. I., and Adler, B. C.: External Otitis: Cytologic Study of Secretions, Laryngoscope **60**:543-550 (June) 1950.

2. Bjuggren, G., and Tunevall, G.: The Pfeiffer Bacillus in Otitis in Children, Acta oto-laryng. **38**:130-146 (April) 1950.

Nielsen [3] summarizes the bacteriological findings of the Norwegian Symposium on penicillin. The clinical results of treatment with penicillin closely followed the results indicated by the sensitization tests. In his series of 79 cases of meningitis occurring in acute otitis, 45% of the infections were caused by pneumococci, 11% by hemolytic streptococci, and 2% by nonhemolytic streptococci; in 30% there was no growth on culture. In meningitis occurring in chronic otitis, pneumococci were found in 15% of cases and nonhemolytic streptococci in 7.5%.

ANATOMY

Druss,[4] in examining histological sections of the temporal bone, found several cases in which there was extension of infection into the carotid canal. In one case there was definite necrosis of the bony wall and accumulation of enough purulent infiltration to narrow the carotid artery by approximately 50%. This occurred in a case of petrositis. In a case of thrombosis of the jugular bulb there was necrosis of the bony plate between the bulb and the carotid artery. [It is possible that this type of infection might account for the occasional reported case of arteriovenous fistula sometimes seen in this region. The illustrations are excellent.]

CHEMOTHERAPY AND BIOTHERAPY

Jersild and Kiørboe [5] treated and report on 266 patients with otitis media, the majority of whom received penicillin twice daily. It was at first intended that every other patient should receive no penicillin, but it was soon found that the benefit of penicillin was so great that the use of control cases was stopped and penicillin given to all patients. There were 47 control patients, all with scarlet-fever otitis, "with 66 ears attacked." In a group of 97 scarlet-fever patients receiving penicillin, 144 ears were attacked. There were 122 other patients with otitis media who received penicillin. In the control group the average duration of discharge was 36.5 days, while in patients receiving penicillin it averaged 6.5 days. Hemolytic streptococci could be cultured from the control group for an average of 20 days but for only one day in the group treated with penicillin. In the control group mastoid operations were necessary in 30.3%, but in the penicillin-treated group only 3.5% needed operation. Only 45% of the patients could be made to return for examination at the end of three months, and it was found that 24% had relapses, the highest per cent occurring in children 3 years old or younger.

Friedman [6] reports the case of a woman of 52 in whom in addition to diabetes there developed otitis media from which Klebsiella pneumoniae was cultured. She

3. Lund, R.; Dohlman, G.; Holmgren, S.; Nielsen, J. C.; Permin, P.; Poulsen, I. P. V.; Rasmussen, H., and Riskaer, N.: Penicillin in Severe Otorhinolaryngological Complications: A Symposium, Copenhagen, Danish Otolaryngological Society, 1950.

4. Druss, J. G.: The Carotid Canal as a Pathway for Extension of Infection in the Temporal Bone, Ann. Otol. Rhin. & Laryng. **59**:166-178 (March) 1950.

5. Jersild, T., and Kiørboe, F.: Effect of Discontinuous Penicillin Therapy in Acute Suppurative Otitis, with Special Reference to Otitis in Children, Acta oto-laryng. **38**:8-17 (Feb.) 1950.

6. Friedman, E. A.: Acute Otitis Media Due to Klebsiella Pneumoniae Treated with Chloramphenicol (Chloromycetin®), Arch. Otolaryng. **51**:420-421 (March) 1950.

did not improve with penicillin. A sensitization test was done, and it was found that the organism was sensitive to chloramphenicol (chloromycetin®) and not to the other antibiotics. Recovery was prompt when chloramphenicol was given.

Lederer [7] warns the otologist not to trust the antibiotics too completely and thus fail to make the exact diagnoses that tend to lead to better treatment. The best protection against future chronicity and deafness is still a well-performed incision of the tympanic membrane. Then, and then only, should antimicrobial therapy be used. [Most acutely infected ears respond to penicillin without incision of the drum membrane. I think it safe in most cases to delay incision a day or two while observing the patient's response.]

Dohlman and Holmgren [8] comment on allergic and toxic effects of penicillin given intrathecally. It has been reported by others that high concentrations of penicillin are extremely irritating to the central nervous system when administered intrathecally. In 420 cases there were 65 with symptoms probably indicating irritation. One patient, a 26-year-old man, was given a single dose of 200,000 units, which was followed by convulsions and death on the next day. There were 12 patients in whom convulsions developed; 5 of them died. He concluded that the dosage should not exceed 10,000 units twice daily for the first two days of treatment only. Increase in the intramuscular dose might eliminate the need for intrathecal administration. He advises large doses of barbiturates if convulsions occur.

Merei [8] noted many reactions of nausea, headache, and fever after intrathecal administration of penicillin for meningitis. After noting the sudden disappearance of these symptoms with the stopping of penicillin administration, he gave penicillin by lumbar and cisternal punctures to 23 patients who had normal temperatures but who were under examination for epilepsy or schizophrenia. About half of them had reactions of headache, increased cell count, and other symptoms of meningeal irritation. In two years he treated 31 patients with meningitis with intrathecal injections of penicillin and did not note these reactions during the acute stages of the disease. After variation of the dose he decided that 10,000 units per cubic centimeter of diluent, cerebrospinal fluid or isotonic sodium chloride solution, were the least irritating solutions.

Richtner [9] treated 565 patients with acute otitis media with sulfonamide preparations. Cultures were made and the organisms were tested for sensitivity to sulfonamides and penicillin. The majority of the organisms were not sensitive to the sulfonamides by test, and even the patients with sulfonamide-sensitive organisms did not seem to improve with administration of sulfathiazole, sulfadiamine (5-amino-2-sulfamyl-benzoic acid), or "sulfamethin."

Titche [10] observed 36 patients with tuberculous otitis media during treatment with streptomycin. Of the 32 patients receiving the drug parenterally, 51% were

7. Lederer, F. L.: The Maginot Line of Otology: Dependence on Antibiotics in Suppurative Middle Ear Disease; Facts and Fancies, Arch. Otolaryng. **52**:533-537 (Oct.) 1950.

8. Merei, L.: Reactive Symptoms After the Intrathecal Administration of Penicillin, Laryngoscope **60**:581-592 (June) 1950.

9. Richtner, N. G.: Experiences of Sulfa Treatment of Acute Otitis in an Out Patient Series from the Ear Department of Södersjukhuset, Acta oto-laryng. **38**:453-459 (Oct.) 1950.

10. Titche, L. L.: Streptomycin in Treatment of Tuberculous Otitis Media, Arch. Otolaryng. **51**:826-830 (June) 1950.

healed, 31% improved, and 17% unimproved. Four patients were treated by displacement technique, using 1 gm. of streptomycin in 1 cc. of water. All four patients were cured; in one, the ear became dry after the third treatment.

Kersley [11] treated 35 patients with chronic otitis media by systemic administration of streptomycin, with excellent results. He was careful to rule out patients with cholesteatoma or bone necrosis who obviously needed surgery. Most of his patients were given 0.5 gm. of streptomycin three times daily for about 10 days. In addition, the ears were cleaned three times daily and ear drops of 5 to 10% streptomycin were instilled. In 3 of the 35 patients toxic reactions developed. Cultures were made in all of the cases, and the predominant organisms were found to be sensitive to streptomycin, and all except one were insensitive to penicillin. In 11 cases Proteus vulgaris was cultured, in most of them in pure culture. In 60% of the cases the ear became and remained dry, and in most of the remaining 40% the disease became "moist and inactive." Kersley concluded that streptomycin is "a remarkable advance in our attack on this lethal and revolting disease."

Huggill and Steel [12] report that in after-treatment of mastoid and fenestration cavities the very frequent infections have been very hard to control with the usual antiseptics. The detergent antiseptics seem to encourage the growth of Pseudomonas aeruginosa (B. pyocyaneus). They advise careful search for granulations and use trichloroacetic acid in their destruction. "The only [antibiotic] found to give really satisfactory results in the majority of cases was Streptomycin; however . . . some strains of B. pyocyaneus are resistant to this drug." The cavity was cleaned and dried, and a 1% streptomycin powder in powdered lactose was blown in with a powder blower.

[Streptomycin powder diluted with 5 parts of boric acid has been used now for three years in these cases, also in external otitis, and has been found most satisfactory in my hands and in the hands of several of my colleagues. Sensitizations seem to occur in about 10% of the cases; therefore in mild infections it is wiser to try a milder remedy, such as the time-tried boric acid and iodine powder, or, where an ointment can be used, bacitracin, whose incidence of sensitizations is lower.]

OTITIS WITH EFFUSION

Hoople [13] has written an article stressing the frequency with which otitis media with effusion is missed in diagnosis. He believes that the chronic or recurring cases arise from blockage of the Eustachian tube but does not know why some blocked ears fail to fill with serum. The diagnosis is made from the complaint of painless impairment of hearing in cases in which there is an amber color of the drum membrane, usually without a fluid level. The diagnosis is confirmed by inflation of the tube or incision of the drum. [Otologists not familiar with this condition should read this article in full.]

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11. Kersley, J. A.: Uses of Streptomycin in the Treatment of Chronic Suppurative Otitis Media, J. Laryng. & Otol. **64**:258-268 (May) 1950.

12. Huggill, P. H., and Steel, A. E.: Infections of the Mastoid Cavity, J. Laryng. & Otol. **64**:645-646 (Oct.) 1950.

13. Hoople, G. D.: Otitis Media with Effusion: A Challenge to Otolaryngology, Laryngoscope **60**:315-329 (April) 1950.

ACUTE OTITIS MEDIA AND MASTOIDITIS

Poulsen [14] did follow-up hearing examinations on 210 children several years after they had had otitis media. He found a higher per cent of hearing loss than is usually expected and found that the amount of loss became greater the longer the duration of the infection.

In 141 cases of nonrecurring otitis media, 73% of the patients had no hearing loss. In 23% there was a loss in the higher notes and in 11%, a loss for the medium and low notes, patients with loss in the lower tones generally being found among those with protracted otitis. Of 91 patients with recurring otitis media, 9% had high-tone loss, 50% had losses for the low or for all the tones, and 41% had normal hearing.

Of the 74 patients who had simple mastoidectomy, 57% had normal hearing, 12% had losses in the high tones, and 31% had losses for the lower tones or all tones. Poulsen found that "restitution of hearing was the better the earlier in the course of the affection the mastoidectomy was performed."

Pohlman,[15] in an article on otitis media in infants, suggests that the presence of mesenchyme (embryonic tissue) in the middle ears of children may be an important factor in their susceptibility to otitis media. The normal infant shows no mesenchyme in the middle ear at birth. Since there is a definite familial tendency toward otitis media, children in such families should have prophylactic treatment by careful removal of tonsils and adenoids and possibly by radiation therapy. Pohlman prefers x-ray treatment to use of radium applicators for younger children. He advises against indiscriminate use of radium in the nasopharynx and does not use it at the time of tonsillectomy. He concludes that the preservation of hearing should be the otologists' first consideration in management of otitis media and antibiotics should be used freely with this in mind.

Poulsen [16] of Copenhagen took roentgenograms of 210 children to determine the development of the cellular system of the temporal bone after otitis media. The children were all over 5 years of age when roentgenograms were taken and all under 3 years when they had otitis media. Among the children who had no recurrence of otitis media, the pneumatization proceeded normally in almost all cases. In those with repeated and prolonged infections, about 25% were found to have arrested pneumatization. The degree of inhibition was proportional to the duration and number of attacks.

CHRONIC OTITIS MEDIA

Jelnes [17] (Denmark) examined 50 patients before and after radical mastoidectomy to determine the effect on the hearing. Careful audiograms showed no change in 62% and poorer hearing in 36%. There was one case in which the hearing

14. Poulsen, I. P. V.: Otitis Media Acuta in Children, 0-3 Years of Age, Acta oto-laryng. **38**:120-129 (April) 1950.

15. Pohlman, M. E.: Otitis Media in Children, Ann. West. Med. & Surg. **4**:626-628 (Oct.) 1950.

16. Poulsen, I. P. V.: The Pneumatization of the Mastoid Bone After Otitis Media, Acta oto-laryng. **38**:511-516 (Dec.) 1950.

17. Jelnes, K. T.: The State of Hearing Before and After Radical Operation for Suppurative Otitis Media, Acta oto-laryng. **38**:216-224 (June) 1950.

was improved. He presents carefully made charts to show the loss in each of the different octaves. The greater losses occurred in the lower tones. The greatest loss occurred, of course, in the recent cases with good preoperative hearing. The average loss for conversational tones in the postoperative group was about 50 db.

Birrell [18] (Scotland) writes of the difficulty in handling chronic draining ears in children. He advises extending the usual postaural mastoidectomy forward to clean out the attic thoroughly. He prefers to leave the posterior bony wall intact. In cases of cholesteatoma he advises the modified radical technique. In 30 children followed for more than a year after operation he obtained dry ears in 73% within four weeks. There were no complications in the series. The hearing was improved in all the children with dry ears.

Young,[19] in his presidential address before the Otology Section of the Royal Society of Medicine, gave a very interesting review of the theories of development of cholesteatoma. He prefers the word "cholesteatosis" since it has the Greek ending "-osis" suggesting "containing" rather than "-oma" suggesting "tumor." He cites D. McKenzie and A. Tumarkin, who doubted the theory of the migration of epithelium from the skin of the canal through marginal perforations but believed that the epithelium of the attic, being squamous, could under certain conditions desquamate and produce cholesteatosis. He mentions also that lack of pneumatization is so frequently associated with cholesteatosis that it may be considered a possible cause of cholesteatosis. In closing he cites the case of a 23-year-old nurse who became suddenly hard of hearing in one ear. There had been no history of ear trouble except for an earache in childhood. Later a minute perforation in the attic area developed. Pain was first noticed five months after impairment of hearing. Roentgenographic examination showed a sclerotic mastoid, and in the operation a small cholesteatoma in the attic and antrum was revealed. In the discussion that followed, Watson-Williams said that he found it impossible to believe that a pneumatized mastoid could later become sclerotic. [Several years ago McMillan reported the case of a nurse at the Massachusetts General Hospital in whom mastoiditis developed in a pneumatic mastoid. The infection subsided without operation, and six months later roentgenograms showed the mastoid to have changed to a sclerotic type.]

Hallpike in continued discussion of Young's paper said that there were cases of cholesteatoma arising from the temporal bone that had nothing to do with the middle ear and showed no evidence of infection. These areas had been found in the course of histological preparations. He said that he thought these proliferative masses were caused by irritation of infection in childhood and were all secondary cholesteatoma. [This may be a reference to small cyst-like collections of cholesterol crystals sometimes found in the middle ear without clinical cholesteatoma.]

McGuckin [20] (England) in a discussion of attic suppuration stated that cholesteatoma is not a separate entity but merely one tissue reaction of the epitympanic

18. Birrell, J. F.: An Extended Schwartze (Cortical) Mastoid Operation in the Treatment of Chronic Mastoiditis in Children, J. Laryng. & Otol. **64**:554-560 (Sept.) 1950.

19. Young, G.: Aural Cholesteatoma, or Cholesteatosis: A Review, J. Laryng. & Otol. **64**:271-278 (May) 1950.

20. McGuckin, F.: Attic Suppuration, J. Laryng. & Otol. **64**:693-699 (Nov.) 1950.

lining to some past sepsis or irritant. Attic disease may be insidious and be already an established chronic lesion when first manifest. It may proceed to complete resolution without subjective complaint. He concludes that the word "cholesteatoma" must still be used for clinical purposes, but states that he has become more catholic than Tumarkin by "abolishing the cholesteatoma doctrine altogether . . . Why assume there are two varieties of cholesteatoma when none will do?"

Tumarkin [21] (Liverpool) reports again on his "premeatal attico-antrotomy" and presents another series of illustrations. Two of the plates are stereoscopic photographs. He states that his attitude toward attic disease has altered in the past five years. He originally performed a cleaning out of the attic only. Now he always exposes the antrum. He believes that there are more cases of "enclosed epitympanitis" since the advent of the antibiotics and that many cases of obscure deafness result from these infections without drainage. He reports five illustrative cases. One of them was in a man seen at the request of his neurosurgical colleagues. The man had an intracranial lesion. The pars tensa was absolutely normal except for a tiny indolent polyp perforating the pars flaccida. There was a little whitish debris but no obvious discharge or pulsation. "With some real reluctance, I opened his mastoid and to my amazement found a completely gangrenous lateral sinus and an extradural abscess extending to the torcula. At a later operation, a temporosphenoidal abscess containing nearly an ounce of pus was evacuated." [Most of the operators who have tried Tumarkin's technique report a lack of space and prefer the wider endaural incision.]

Seed [22] (England), in another paper on attic suppuration, emphasizes the need of studying these sometimes minute perforations with the magnifying electric otoscope. In teaching his students he tried to get them to recognize the dangerous ear with the cholesteatomatous response from squamous epithelium and the nondangerous mucopurulent response of ciliated epithelium. He felt that Tumarkin's premeatal operation would not always leave a meatus large enough for future inspection and treatment. He prefers the endaural approach and recommends the excision of a triangle of temporal fascia as described by House.

In the interesting discussion that follows these four papers, Watson-Williams states that he had been astonished on looking up his notes to realize that in only seven cases in 24 years had he found at operation the disease limited to the attic only; in the majority the pathological condition involved the antrum and part of the mastoid. T. A. Clarke said that he believed that once the attic had been opened, the incus should always be removed; otherwise it would remain a useless hindrance to hearing. This was questioned by others present. [This symposium and the discussion which followed should be read in the original.]

Barley [23] (England) reports a massive cholesteatoma which extended from the tip of the mastoid to the root of the zygoma in a child of 3 years. The duration of drainage was nine months. The drainage had begun without pain, and there was

21. Tumarkin, A.: Attic Suppuration, J. Laryng. & Otol. **64**:611-624 (Oct.) 1950.

22. Seed, G. S.: Attic Suppuration, J. Laryng. & Otol. **64**:700-707 (Nov.) 1950.

23. Barley, D. A.: A Case of Massive Cholesteatoma in Childhood, J. Laryng. & Otol. **60**:19-20 (Jan.) 1950.

a foul odor shortly after onset. Both mastoids were extensively pneumatized. It was thought that the mass probably arose from a developmental embryonic rest rather than from extension from an otitis media. Roentgenograms suggested a similar process in the opposite mastoid, but an exploratory mastoidectomy showed no pathologic changes.

Asherson [24] (London) reports the startling improvement in hearing observed in deafened persons when an insert of cotton saturated in liquid petrolatum can be placed over the region of the round window. Cases suitable for trial are those in which the patients have had radical mastoidectomy or have a large tympanic perforation. Asherson found that over 60% of patients with radical mastoid cavities would have some improvement with the insert. These inserts can be worn for 6 to 10 weeks without changing. Most patients can be trained to readjust or to replace the insert. To be a suitable patient one must have severe deafness, exceeding 30 db., which must be bilateral and must result from suppuration. The deafness must not exceed 70 db. He raises the question whether it might not be advisable to try removal of enough of an old scarred drum membrane to allow the insert to reach the region of the round window.

EXTERNAL OTITIS

Sullivan and Smith [25] give directions for the office treatment of external otitis and otitis media. They have observed so many local reactions to penicillin, the sulfonamide compounds, streptomycin, and substances such as dibromsalicylaldehyde that they use these substances with great caution in treatment of infections that do not clear with careful cleansing and application of mild antiseptics, such as methylrosaniline chloride (gentian violet). They are using 1% acetic acid, alternating with an alkali in some cases, with good results.

Ochs,[26] in an article on the treatment of external otitis, states that he has been using 2% acetic acid and dilute aluminum acetate for three years with excellent results. He has treated 165 patients with 248 infected ears "without a single failure to clear up the acute phase of this condition. In every instance the discharge stopped within hours . . . the foul odor of infected macerated skin was lost and the swelling subsided promptly." After the acute phase the patient is given an ointment containing 3% sulfur and 3% salicylic acid. Three typical cases are given.

Ochs [27] used household vinegar in treating external otitis and had such good results that he tried vinegar in chronic otitis media. Thirty-eight patients with 44 chronic middle ear infections were thus treated; only 8 patients failed to have clearing, and it was decided that in these cases the infection was too deep to be reached or that bony necrosis was present. Displacement treatment was used, and 30 patients had healing with three or fewer treatments. Ochs then reviewed the literature

24. Asherson, N.: Deafness Due to Chronic Otorrhœa: Alleviated by Tympanic Insert, J. Laryng. & Otol. **64**:239-248 (May) 1950.

25. Sullivan, J. A., and Smith, J. B.: The Office Treatment of Otitis Media, Ann. Otolaryng. **59**:364-380 (June) 1950.

26. Ochs, I. L.: Treatment of External Otitis: A Simple and Effective Technique, J. A. M. A. **142**:1361 (April 29) 1950.

27. Ochs, I. L.: Use of Vinegar as an Antibiotic in the Treatment of Chronic Middle Ear Disease, Arch. Otolaryng. **52**:935-941 (Dec.) 1950.

and found numerous reports of the effectiveness of acetic acid for both Gram-negative and Gram-positive organisms. Many of the patients had been treated without success with the sulfonamides and antibiotics. Ochs decided that vinegar must have antibiotic qualities.

Wright [28] did careful sensitization tests on cultures from 23 ears with acute or chronic external otitis and reported excellent results from treatment with aureomycin. Pseudomonas aeruginosa and Micrococcus pyogenes var. albus (Staphylococcus albus) were the two organisms most commonly found. Fungi were not found in any of his cases. The technique of the sensitization tests is simple, and such tests could easily be done by any laboratory. Forty-four patients with external otitis were treated.

In mild cases he found simple cleaning of the ears and the use of ear drops containing 1% of salicylic acid in 70% alcohol were satisfactory.

In severe cases the use of aureomycin powder after cleaning of the canal was most effective. He advised using aureomycin full strength in an Upjohn penicillin inhaler. He reported "considerable trouble with allergic drug reactions and with recurrences in spite of the apparent sensitivity of the low virulence organisms." Sulfathiazole and sulfamylon® showed such poor results on sensitivity tests that they were almost never employed. Four cases were reported in detail.

Haley,[29] of the Bacteriological Department of Duke Hospital, wrote four papers on the problem of otomycosis. In the first paper she reported culturing of material from 216 normal ear canals and 367 infected ears for fungi. About as high a percentage of positive cultures was obtained from normal ear canals as from infected ears. Then she cultured 106 normal canals and 199 "ear trouble" cases for bacteria. The normal canals cultured out many staphylococci and other bacteria but no Pseudomonas organisms. An attempt was then made to infect the ears of rabbits with fungi. It was necessary to traumatize the ears before an infection could be produced. A typical case of otomycosis could not be produced. Twelve patients with the diagnosis of otomycosis were then given skin tests for fungi, and none of the tests produced positive reactions. Cultures from four of them yielded Aspergillus niger, but the majority of the cultures grew staphylococci and Pseudomonas organisms. Haley believes that the external otitis probably represents localized tissue sensitivity. [The conclusion of this study agrees with the opinion of many other careful workers, that the cause of external otitis is rarely fungi.]

McLaurin [30] treated 122 patients with 164 infections, using sulfamylon,® a sulfonamide derivative. There were 65 patients with otitis externa, 34 with chronic otitis media, 12 with infected radical mastoid cavities, and 11 with infected fenestration cavities. In this series only six patients paid more than three visits to the office. There were no sensitization reactions. McLaurin found that the Gram-negative organisms responded best, and if Gram-positive organisms were found, penicillin or nitrofurazone (furacin®) was used. He urged care in cleaning and

28. Wright, W. K.: Use of Aureomycin in External Otitis, Arch. Otolaryng. **52**:74-81 (July) 1950.

29. Haley, L. D.: Etiology of Otomycosis, Arch. Otolaryng. **52**:202-224 (Aug.) 1950.

30. McLaurin, J. W.: The Local Application of "Sulfamylon" [Para-(Aminoethyl)-Benzene Sulfonamide Hydrochloride] in Otitis Externa and Chronic Otitis Media, Laryngoscope **60**:480-488 (May) 1950.

drying the ears before using any medication. If the patient did not show improvement on the second visit, cultures were made and another medication used if indicated.

MENINGITIS

Rasmussen summarizes the results in 203 cases of meningitis in the Danish, Swedish, and Norwegian symposium.[3] All patients were treated with penicillin, and most of them also received sulfonamide preparations. The mortality was 20.7% for the whole group, 33% in 36 cases of pneumococcal meningitis, and 31% in 15 cases of streptococcal meningitis. A comparison between patients treated with intramuscular injections alone and patients treated both intramuscularly and intrathecally shows that the mortality in the two groups is about equal. This indicates that intrathecal administration of penicillin is not necessary in all cases. There were 36 cases in which meningitis developed in the first six days of acute otitis; in 24 cases, the patients were operated on, and in 12, they were not. There were 5 deaths among the 24 patients operated on and 4 deaths among the 12 not operated on. His general rule was to perform mastoidectomy in all cases of otitic meningitis of more than one week's duration.

Dohlman and Holmgren, in the Danish, Norwegian, and Swedish symposium,[3] comment on the allergic and toxic effects of penicillin given intrathecally. It has been reported by others that high concentrations of penicillin are extremely irritating to the central nervous system when administered intrathecally. In 420 cases there were 65 patients with symptoms probably indicating irritation. One patient, a 26-year-old man, was given a single dose of 200,000 units which was followed by convulsions and death on the next day. There were altogether 12 patients in whom convulsions developed, and there were 5 deaths during convulsions. It was concluded that the dosage should not exceed 10,000 units twice daily for the first two days of treatment only. Increase in the intramuscular dose might eliminate the need for intrathecal administration. The authors advise large doses of barbiturates if convulsions occur. [This is an interesting contribution.]

Merei [31] of Budapest reports nine cases of recurring meningitis and discusses the treatment. He concludes that the early type of meningitis that follows otitis media of only a few days' duration will usually clear up without operation, but if there is a recurrence it is necessary to search for and drain the focus. If an operation has already been done, the wound should be revised if no other focus can be found. Merei continues intrathecal administration of penicillin until the cell count of the spinal fluid is not more than 100 per cubic centimeter or until reactive symptoms evoked by penicillin occur. Systemic administration of penicillin is then continued for two or three weeks, perhaps with sulfonamides.

Taylor [32] reports three cases of chronic mastoiditis due to Proteus vulgaris (B. proteus). Two of the patients had symptoms indicating meningitis, and the third, a child of 13, had epileptiform attacks of one day's duration, which cleared up after a radical mastoidectomy was done. The dura and the lateral sinus were covered with granulations. There were no more attacks after the pathological con-

31. Merei, L.: Recurring Meningitis, Acta oto-laryng. **38**:201-215 (June) 1950.

32. Taylor, L. R. S.: Bacillus Proteus Mastoiditis with Recovery, J. Laryng. & Otol. **64**:82-84 (Feb.) 1950

dition was removed. Penicillin was given this patient, while the other two patients with meningitis received large doses of penicillin plus streptomycin.

Mastoiditis caused by P. vulgaris is rare, is likely to be resistant to treatment, and is frequently complicated by intracranial lesions. In his review of the literature, Taylor found only 28 cases reported. In the second case the patient had lateral sinus thrombosis; he recovered without ligation of the jugular vein. In all three cases the patients were cured.

Merei and Kristof [33] report two cases of tuberculous meningitis due to tuberculous otitis media. Streptomycin not being available, both patients died. The authors urge early mastoidectomy in cases of otitis media occurring in tuberculous patients. [This view would be modified in view of the success now obtained with streptomycin. Tuberculous meningitis is rarely due to direct extension from otitis media.]

Jemmi [34] of Italy reports two cases of fracture involving the temporal bone in which meningitis developed late after the accident. In one case the patient recovered. He points out that healing of such fractures is by fibrous union. This fibrous union is not very resistant to infection, and meningitis may occur years after the accident.

Judge and Davis [35] report the case of a 25-year-old man who had had a foul-smelling chronic otitis media for five years. Two days following the performance of a modified radical mastoidectomy, symptoms of meningitis developed. Cultures from the spinal fluid showed Escherichia coli. Chloramphenicol (chloromycetin®) apparently controlled the infection after penicillin failed to produce improvement. The patient recovered without sequelae.

Weber [36] of Finland treated 19 patients with meningitis with penicillin. There were 15 cases resulting from otitis media and 4 from other causes. Five of the 19 patients died (26.6%). There were 10 cases of pneumococcal meningitis, and in them there was a 50% mortality. However, three of the five deaths were in late cases and occurred within 48 hours following the patients' admission.

Weber considered suboccipital administration of penicillin too difficult and gave intraventricular injections of penicillin through a trephine opening if the patient did not respond to penicillin given by the usual lumbar puncture. He strongly advises drainage of the focus and continuation of the therapy for a week or more after the meningeal symptoms have subsided. [These are well-summarized cases and deserve study.]

THROMBOSIS OF THE LATERAL SINUS

Smith [37] of Edinburgh mentions the change that the antibiotics have made in the complications of otitis media, especially in thrombosis of the lateral sinus. He

33. Merei, L., and Kristof, I.: Otogenous Tuberculous Meningitis in Infants, Acta oto-laryng. **38**:40-44, 1950.

34. Jemmi, C.: Delayed Meningitis Following Fracture of the Temporal Bone, Oto-rino-laring. ital. **17**:15, 1948.

35. Judge, A. F., and Davis, W. L.: Otogenic Meningitis Due to Escherichia Coli: Treatment with Chloromycetin, Laryngoscope **60**:186-192 (Feb.) 1950.

36. Weber, C.: Treatment of Otorhinogenic Meningitis with Penicillin, Acta oto-laryng. **38**:167-178 (April) 1950.

37. Smith, A. B.: Otogenous Thrombophlebitis of the Lateral Sinus and Jugular Bulb, J. Laryng. & Otol. **64**:12-16 (Jan.) 1950.

has seen 30 cases in his practice, 8 of which were seen since the introduction of antibiotics. In the 22 cases observed before penicillin was available, there were seven deaths, a mortality of 32%. Of the seven fatal cases, two arose from acute otitis media and five from chronic otitis media. A history of chills was obtained in only three cases. In considering the eight cases for which penicillin was available, Smith states that all of them occurred in chronic otitis media, and in six the patients were under the age of 12 years. All of them recovered after operation, none requiring ligation of the jugular vein. He states that thrombosis of the lateral sinus, as a complication of acute otitis, has largely disappeared and that, if it does occur, it will probably be controlled without a diagnosis. Smith concludes that early operation on thrombosis of the lateral sinus is still important and that the best guide to the necessity of operation is the temperature chart.

Merei [38] of Budapest reports a case of necrosis of the wall of the sigmoid sinus and jugular vein occurring in a man of 25. The lateral sinus thrombosis had extended downward, forming a large abscess that filled the jugular vein down below the clavicle. Most of the anterior wall of the jugular vein had been destroyed. A lung abscess formed about the 14th day of hospitalization, in spite of the fact that an operation had been done on the day after admission and in spite of penicillin and sulfamethylthiazole administration, which had started 10 days before admission to the hospital. The onset of high fever was five weeks before admission. Swelling and tenderness in the neck were noticed four days before admission. The patient recovered.

Morgenthal [39] of South Africa reports the rare case of a girl, 16, who had had a discharge from her left ear. Occipital pain, dizziness, vomiting, and fever developed, the acute symptoms developing two weeks before operation. During the radical mastoidectomy, it was found that the sigmoid sinus had thrombosed and ruptured. By packing off the sinus and giving penicillin, a cure was obtained without ligation of the jugular vein.

Poulsen [3] reports on 25 cases of lateral sinus thrombosis in which there were three deaths. In two of the fatal cases there was also meningitis and in the third a temporal lobe abscess. In ten of the cases there was ligation of the jugular vein, but Poulsen feels (with the majority of others) that with the sulfonamides and penicillin it should almost never be necessary to ligate the jugular vein.

LABYRINTHITIS

Lund [3] summarizes the findings in 101 cases of labyrinthitis. There were 42 cases of serous labyrinthitis, 55 cases of the acute diffuse destructive type, 3 cases of circumscribed labyrinthitis, and one of the chronic diffuse destructive type. He concluded that "in cases (of labyrinthitis) commencing with acute middle ear suppuration, labyrinthectomy is almost always unnecessary and, possibly, even harmful. Only in very rare cases which prove refractory to chemotherapy and antibiotic therapy should labyrinthectomy be considered."

38. Merei, L.: Neurosis of the Wall of the Sigmoid Sinus and the Jugular Vein: Lung Abscess; Recovery, Acta oto-laryng. **38**:78-81, 1950.

39. Morgenthal, J. W.: Spontaneous Rupture of the Sigmoid Sinus in a Case of Mastoiditis, J. Laryng. & Otol. **64**:521-522 (Aug.) 1950.

BRAIN ABSCESS

Harpman [40] (England) reports his observations on the diagnosis of brain abscess and the treatment of 18 patients. His review of the literature comprises 138 references. Among other conclusions, he advises that brain abscesses should be opened early, without delay, to allow encapsulation. Pathological conditions in the ear should be removed before searching for the abscess, since exposure of the dura during the mastoidectomy frequently reveals a tract which leads to the abscess. [His discussions on technique and after-care deserve study.]

Brunner [41] reports that he has observed 35 otogenous temporal lobe abscesses and reports 3 cases in detail. He advises that the approach be made through the mastoid rather than through a separate incision above. In spite of the disadvantage of herniation of the brain, he makes a cross incision through the dura after locating the abscess with a cannula. Then a part of the capsule of the abscess is cut out for drainage. This technique he calls the open method. In six patients treated by this technique there was one death.

Riskaer [3] reports in the Danish, Swedish, and Norwegian synposium on 14 cases of brain abscess. There was a 50% mortality. He blames this high mortality on the fact that several of the patients came in too late for the treatment to be effective. He states that the prechemotherapy mortality of 86 to 55% (recorded by several observers) has been reduced to no more than 20% in several clinics since the introduction of penicillin combined with the sulfonamides. The surgical treatment should be puncture, drainage, or extirpation, according to the nature of the case. Riskaer cites Le Beau, who did extirpation en masse in 14 cases of cerebral abscess without losing a patient. Before the use of penicillin, patients in three similar cases died. Penicillin was given intrathecally in all cases in which the organism showed sensitivity to penicillin.

PETROSITIS

Permin [3] summarizes eight cases of petrositis. There was only one death, that of a 10-month-old boy who was given less penicillin than is now advised.

ENCEPHALITIS

Möller [42] of Sweden reports a case of contralateral hemiplegia occurring in a 39-year-old woman as a complication of otitis media. Hemiplegia appeared first on the same side as the otitis media. This disappeared, and hemiplegia appeared on the opposite side. Because of normal spinal fluid, only slight rise of temperature, and other symptoms, it was decided that the encephalomyelitis was probably of the postvaccinal virus type. The patient recovered completely except for symptoms of depression and sleeplessness which persisted for many months.

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40. Harpman, J. A.: On the Treatment of Otogenic Brain Abscesses, J. Laryng. & Otol. **64**:319-334 (June) 1950.

41. Brunner, H.: Treatment of Otogenous Temporal Lobe Abscess, Ann. Otol. Rhin. & Laryng. **59**:943-957 (Dec.) 1950.

42. Möller, F.: Nervous Complications Arising from Otitis Media, A. M. A. Arch. Otolaryng. **38**:74-77 (Feb.) 1951.

TECHNIQUE

Lempert [43] traces the evolution of the endaural mastoid operation and, as usual, has illustrated his article with excellent drawings showing the different steps in his technique. [This article and the illustrations deserve study by anyone interested in temporal bone surgery. It does not lend itself well to condensation.]

McQuiston [44] presents a modification of the endaural incision for chronic mastoiditis, in which he removes most of the skin of the external canal and uses it as a skin graft. He then follows the technique, as described by Lempert and others, of opening the antrum by going gradually through the posterosuperior canal wall. He insists on careful postoperative watching of the cavity with curettement of any granulations that may form.

Popper [45] of South Africa presents more illustrations of his premeatal incision and technique which he uses in his fenestration and chronic mastoid surgery. [An interesting technique. Most of the operators who try it feel that they do not have quite enough room to work properly.]

Sheridan [46] describes a modified radical mastoidectomy which he does through the postauricular incision. He removes the bridge, incus, and head of the malleus but does not leave any opening in the meatus even if cholesteatoma is present. In only one case out of a series of 33 was it necessary to do a complete radical mastoidectomy later. The posterior and superior wall of the membranous meatus is cut across to take off tension when the retractor is used; "otherwise there is a tendency later for detachment to take place at or near the tympanic ring." This incision heals without sutures, and no stricture of the canal is caused by it. Prolonged observation showed that cholesteatoma did not tend to re-form. In 15 of the 33 cases there was improvement of the hearing as measured by voice tests, since "postoperative audiograms require more time than I or the patients have been able to give." There are brief abstracts of the 33 cases, most of which showed extreme pathologic changes.

MISCELLANEOUS

Nákó and Hajdu [47] of Budapest reports the unusual case of a man of 50 in whom development of a squamous cell carcinoma in the petrous apex followed a chronic otitis media of 43 years' duration. There was extensive destruction of the petrous pyramid, and a fistula opened into the nasopharynx. An interesting feature of the case was the spontaneous formation of a fistula through the parotid gland seven years before the patient came to the hospital with symptoms of the carcinoma.

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43. Lempert, J.: Modern Temporal Bone Surgery: History of Its Evolution, Laryngoscope **60**:740-778 (Aug.) 1950.

44. McQuiston, R. J.: Endaural Radical Mastoidectomy for Chronic Mastoiditis, Arch. Otolaryng. **51**:596-607 (April) 1950.

45. Popper, O.: Transtympanic Technic in Surgical Otology, Arch. Otolaryng. **51**:356-370 (March) 1950.

46. Sheridan, M. R.: Modified Radical Mastoid Operations with Meatal Preservation and with Observations on Hearing in the Operation Described, J. Laryng. & Otol. **64**:219-232 (May) 1950.

47. Nákó, A., and Hajdu, I.: Extensive Destruction of the Petrous Bone, Acta oto-laryng. **38**:147-154 (April) 1950.

After a radical mastoidectomy, the parotid fistula healed, and there were no symptoms for six years. The author concludes that there was probably no connection between the carcinoma formation and the healed otitis media.

Koch [48] (Stockholm) reports the unusual case of a woman, 61, who had a probable meningocele of the temporal bone. He found only two cases of congenital meningoceles reported in the literature. Koch's patient had suffered repeated attacks of meningitis of uncertain origin. After incision of the drum membrane for acute otitis media, there developed purulent meningitis and a profuse escape of cerebrospinal fluid lasting for 24 hours. Roentgenograms showed a bony defect at the sinodural angle that was assumed to be caused by a meningocele. Penicillin was not then available; so a mastoidectomy was not done. The patient recovered after a few weeks, with normal hearing.

Biggart [49] (Belfast, Ireland) reports a case of mastoiditis with a large extradural abscess in a man of 26 who had no external auditory canal. Roentgenograms showed absence of the bony canal and a sclerotic mastoid. Operation through a posterior incision disclosed a mastoid antrum and aditus, but the middle ear was not explored. The patient was discharged well after three weeks.

960 E. Green St. (1).

48. Koch, H.: Meningocele of the Temporal Bone, Acta oto-laryng. **38**:59-61 (Feb.) 1950.

49. Biggart, H. G.: Mastoiditis in an Under-Developed Ear, J. Laryng. & Otol. **64**:141-142 (March) 1950.

Abstracts from Current Literature

Ear

A Clinical Study of Auditory Damage Following Blows to the Head. Harold F. Schuknecht, Ann. Otol. Rhin. & Laryng. **59**:331 (June) 1950.

Diminished hearing acuity may follow a blow to the head, whether or not a fracture of the temporal bone is demonstrated. Temporal bone fractures may be either longitudinal or transverse to the long axis of the petrous pyramid. When longitudinal, there is bleeding from the ear, rupture of the tympanic membrane, and combined conductive and nerve-type loss of hearing. The conductive deafness is caused by direct injury to middle ear structures and is partially or completely reversible. When the fracture is transverse, there results a severe vertigo with nystagmus to the unaffected side, hemotympanum, complete deafness, and total loss of vestibular function in the affected ear. In this case, the fracture line extends through the inner ear and degeneration of the membranous labyrinth follows.

In patients who show no clinical or roentgenographic evidence of temporal bone fracture after a blow to the head, loss of hearing is of the "nerve" type and is severest for the frequencies above 4096. With severe injury, all frequencies may be involved. This type of deafness frequently is seen in the ear on the side opposite to the temporal bone fracture. The presence of connective tissue and bone in the perilymphatic spaces, with degeneration of Corti's organ and the spiral ganglion, has been demonstrated in the temporal bone removed from patients with deafness following head injury without fracture. Some specimens have shown only severe degeneration of Corti's organ and the spiral ganglion.

Animal experiments demonstrate that hemorrhage into the perilymphatic spaces often occurs after head blows. Degeneration of the hair cells and spiral ganglion was severest in the middle cochlear coil in cats subjected to head blows. It is believed that a traveling pressure wave injures these structures directly.

Seventeen cases of deafness following head injury are included in this report.

Steele, Kansas City, Kan.

Electroencephalography in the Diagnosis of Hearing Loss in the Very Young Child. R. E. Marcus, E. L. Gibbs, and F. A. Gibbs, Dis. Nerv. System **10**:170 (June) 1949.

Education of the hearing-handicapped child, to be effective, must be instituted early, but the diagnosis of profound deafness in the very young is clinically difficult. The authors report an approach utilizing the "arousal response" (waking activity interrupting cortical sleep patterns) in the electroencephalogram of the sleeping child. Various loud, frightening noises were produced, and it was observed that the "arousal response" was obtained when the sound was perceived and was the same as that caused by other types of sensory stimulation. Nineteen patients, from 17 months to 9 years of age, where studied; in 13 no audition could be demonstrated by routine otological testing. Four of the latter group showed some "arousal response." It is concluded that electroencephalography provides a technique for the diagnosis of deafness in the infant and child and that the "arousal response" to appropriate stimuli is an adequate index of hearing. As an additional observation, it was found that of the 13 children with recognized deafness, 7 had electroencephalographic abnormality. The remaining six patients, in whom the primary disorder was retarded speech and an associated incomplete reduction in auditory function, all displayed electroencephalographic irregularities. The relation of hearing loss to disorders of the central nervous system should receive further investigation.

Beaton, Tucson, Ariz. [A. M. A. Arch. Neurol.].

THE MASKING EFFECT OF PENICILLIN IN OTITIC MENINGITIS. ALFREDO MARTINEZ GOYA, ENRIQUE YACUZZI and HECTOR MONACO, Rev. argent. oto-rino-laring. **19**:59 (Jan.-Feb.) 1950.

A case of a 20-year-old aviator with acute otitis media is presented. He was treated with sulfathiazole and penicillin and had an uneventful convalescence. However, three months later he returned to the infirmary with symptoms of pain in his ear, high fever, nuchal rigidity, projectile vomiting, right-sided headache, and mental confusion. The following day he had a spontaneous perforation with a serosanguineous discharge from his right ear. Lumbar puncture revealed an opalescent liquid but no organisms; he had a leucocyte count of 17,200, with 85% neutrophiles. The roentgenogram of the mastoid revealed a rarefaction of the bone. A mastoidectomy had been performed with an extensive exenteration of all the cells. The lateral sinus showed a periphlebitis, but free blood was obtained on puncture and the culture was negative. The dura of the middle fossa appeared normal. The pathological diagnosis of the mastoid bone was chronic osteomyelitis. Postoperatively, the patient was treated with blood transfusions, streptomycin, vitamins, and tonics. His meningeal symptoms disappeared, and he made an uneventful recovery.

Salient features of the case were these: 1. There was acute otitis media which was apparently cured in eight days. 2. The cure was apparently due to a combination of sulfonamides and penicillin therapy. 3. There was a slow progression of the pathological condition which culminated three months later in an acute exacerbation with meningeal symptoms. 4. The causative organism was a Staphylococcus which had become resistant to penicillin but yielded to streptomycin. The latter probably was responsible for complete cure of this patient.

PERSKY, Philadelphia.

STUDIES IN AUDITORY FATIGUE AND ADAPTION. J. D. HOOD, Acta oto-laryng., Supp. 92, 1950.

In 1943 there appeared the "Harvard Study" on deafness and auditory fatigue, the work of H. Davis and his collaborators ("Report on Temporary Deafness Following Exposure to Loud Tones and Noise," Committee on Medical Research of the Office of Scientific Research and Development, Department of Physiology, Harvard, 1943). Not since that time, until the present article, has there been published anything confirming or enlarging upon the subject. This article is a very full and thorough exposition written with clarity of style which makes a rather technical matter easily understood. (This art appears more commonly to be the asset of English authors than of those of any other nationality.)

Davis' advances and discoveries are verified and appear to consist of two types of fatigue which develop from loud sounds—physiological and reversible hearing loss and loss from permanent cochlear damage. The danger point from loud noise is at lower intensities than the loudness producing a tactile or pain response in the ear. Damage pitches give a loss about one-half octave above their pitch, except for the lowest frequencies. Composite noise shows fatigue curves with definite maximum effects at 900, 1800, and 2700 cps. "Stimulus intensity is a vital factor in determining not only extent but also the frequency distribution of pure tone fatigue." The results of the studies are in agreement with the conception that the cochlear ribbon is frequency selective throughout its length and that high tone perception is centered toward its base and low tone perception toward its apex.

The latter half of the study is concerned with a new approach to fatigue which is named "per-stimulation fatigue." It is essentially a testing of fatigue effects by loudness balance, or matching the loudness of sound in the fatigued ear with a fixed loudness in the opposite ear. An intial high fatigue response lasts about one minute, then gradually decreases. This corresponds with results of fatigue tests on other human tissues. This study has confirmed the considerable variation between persons as to fatigability. There is definite information as to spread of loud sounds along the cochlear ribbon. One will learn much of the operations of the cochlea as a receptor from a careful reading of the paper and from this should develop some practical routine fatigue tests for office practice.

MACFARLAN, Philadelphia.

Pharynx

THE RISK OF POLIOMYELITIS AFTER TONSILLECTOMY. GAYLORD W. ANDERSON, GENEVIEVE ANDERSON, AUDRY E. SKAAR, and FRANZISCA SANDLER, Ann. Otol. Rhin. & Laryng. **59**:602 (Sept.) 1950.

A series of 21 cases of poliomyelitis in Minnesota occurring within one month after tonsillectomy during the summer of 1946 is reported. In all, 2,881 cases of poliomyelitis were officially reported in this epidemic. Of these, 2,709 supplied detailed epidemiological data. In the entire series, 226 patients died.

A report is made of 19 of the 21 cases occurring within one month following tonsillectomy. There were three deaths. In 12 patients (63.2%) bulbar poliomyelitis developed, as contrasted with 4 (22.2%) of 18 who had tonsils removed in the second or third month preceding onset of the disease. There was a concentration of cases of bulbar poliomyelitis the onset of which after the operation was within the usual range of incubation periods.

The authors concluded from this study that the risk of poliomyelitis was at least three times as great among patients undergoing tonsillectomy as among a comparable group not undergoing this operation and the risk of bulbar infection was 11 times as great.

STEELE, Kansas City, Kan.

ACUTE LARYNGOTRACHEOBRONCHITIS: AN ANALYSIS OF 1,175 CASES WITH 98 TRACHEOTOMIES. A. REGINALD EVERETT, Laryngoscope **61**:113 (Feb.) 1951.

Everett reviews the cases of 1,175 patients with acute obstructive infections of the larynx, trachea, and bronchi, who were treated at the Willard Parker Hospital between 1940 and 1949. A comparison is made with similar cases occurring between 1930 and 1939. There was a marked decrease in the mortality and in operative intervention, especially intubation, in the 1940 to 1949 series. The use of the antibiotics was largely responsible for this. In addition, in the majority of patients best results were obtained by treatment with a combination of cool water vapor and oxygen.

HITSCHLER, Philadelphia.

ERRORS IN CANCER OF THE LARYNX. JUSTO M. ALONSO, Rev. Otorrinolaring. **9**:113 (Dec.) 1949.

Alonso presents a number of errors encountered in cases of cancer of the larynx. 1. Under errors in treatment he considers the question of independent syphilis or syphilis associated with malignant conditions of the larynx. He feels that in a number of cases, either because of the history or a positive Wassermann reaction, antisyphilitic treatment has been given and, in consequence, valuable time lost before proper treatment for the malignant condition of the larynx has been instituted. 2. Errors in roentgenologic diagnosis are due to faulty interpretation, faulty technique, or a lack of coordination between the roentgenologic and clinical findings. 3. Errors in proper interpretation of biopsies are evident when the pathologic-anatomic diagnosis at times does not coincide with the clinical diagnosis. These errors may at times be due to poor technique in obtaining a proper section for pathological study but oftener are due to a difference of opinion among the pathologists. 4. Errors in proper clinical examination may be the fault of the patient or of circumstances beyond the control of the patient, such as improper visualization of the larynx by either direct or indirect laryngoscopy or by improper clinical study of the patient who may have some general systemic condition directly responsible for the pathological condition of the larynx. 5. Errors in the election of treatment may be made. Judgment must be exercised to determine whether surgical or x-ray therapy or both are indicated. 6. Errors in the extent of surgery or x-ray therapy may be made. The type of surgery indicated should be carefully evaluated on the basis of the type and extent of the pathological condition and whether or not metastasis is present. X-ray therapy in certain cases has been used too sparingly, with the result that there have been recurrences. On the other hand, excessive irradiation has also produced serious after-effects which jeopardize not only the success of any surgical procedure but also the life of the patient.

PERSKY, Philadelphia.

THE RELATION OF HISTOLOGICAL GRADING OF MALIGNANCY AND THE RATE OF RECURRENCES FOLLOWING OPERATION IN LARYNGEAL CANCERS. HIKONOJO IWAMOTO, Jap. J. Otolaryng. **23**:179 (May) 1951.

During the past 15 years, 97 patients with carcinoma were treated with total laryngectomy at Kyushu University Hospital, and 41 had recurrences. Histological grading of recurrent carcinoma according to Broders' classification showed Grade 1—13, Grade 2—39, Grade 3—31, and Grade 4—14. Iwamoto studied the submucous spread of the removed tumors in each group and drew the following conclusions: 1. There was no relation between the degree of malignancy and the age of the patient. 2. Intrinsic carcinoma showed a relatively lower grade of malignancy. 3. The higher the grade of malignancy, the greater was the incidence of cervical metastasis and the more frequent were the instances of recurrence following the operation. 4. The submucous spread of the tumor was proportionately great according to the degree of malignancy, the average being 0.1 mm., 5.4 mm., 6.6 mm., and 5.5 mm. for each grade, respectively. He recommends that the line of incision at the operation be at least 15 mm. beyond tumors visible on the surface of the mucous membrane.

HARA, Los Angeles.

Nose

OBSERVATIONS ON THE LOCAL USE OF CORTISONE IN THE NOSE IN ALLERGIC RHINITIS. J. LEWIS DILL and DONALD S. BOLSTAD, Laryngoscope **61**:415 (May) 1951.

A 1 to 4 dilution of cortisone was used as a nasal spray in 25 patients with allergic nasal trouble. Almost half showed marked improvement, while a little over a fourth failed to improve. In some, particularly those with nasal polyps, there was a recurrence of the trouble after administration of cortisone was discontinued. The work is being continued in the hope of discovering, particularly, how permanent is the effect of such treatment.

HITSCHLER, Philadelphia.

RHINOGENIC BRAIN ABSCESS. C. H. MCCASKEY, Laryngoscope **61**:460 (May) 1951

McCaskey reviews rhinogenous brain abscess and presents an interesting case. Among the subjects discussed are etiology, routes of infection, bacteriology (it is noted that areobic origin favors the formation of a capsule, while anaerobic origin does not), pathological findings, symptoms, roentgenologic findings, prognosis, and treatment. Treating the encapsulated abscess as a tumor and removing it *in toto* gives the best results. It is difficult sometimes to determine just when surgery should be used. If encapsulation occurs, it usually occurs some weeks after the onset of the condition and is usually marked by some diminution in the signs and symptoms. Eradication of the original source of infection is important.

HITSCHLER, Philadelphia.

CEREBRAL SPINAL RHINORRHEA. GERMAN HUGO DICKMANN and ANRES A. VEPPO, Rev. argent. oto-rino-laring. **19**:83 (March-May) 1950.

In discussing cerebrospinal rhinorrhea, Dickmann and Veppo classify this condition as due to (1) cranial fractures, (2) postoperative trauma, (3) spontaneous development, and (4) intracranial tumors. The diagnosis is usually easily established. The principal complication is meningitis. They discuss the various surgical procedures that have been attempted to close the wound. Their own procedure is a modification of William J. German's technique. They usually prefer local anesthesia. They make an osteoplastic flap and expose the dura up to the anterior border of the wound, then raise the frontal lobe and so visualize the granulations of pia mater in the region of the fistula. These are removed with the greatest care to avoid hemorrhage. The olfactory nerve and bulb are exposed up to the perforation of the foramina of the lamina cribrosa. A free muscle graft is inserted, and the surrounding area of the wound is packed with absorbable gelatin sponge. In most cases suturing of the wound is unsatisfactory because of the friability of the dura. On the other hand, when there is a fair-sized laceration the aponeurosis of the temporal muscle is cut to size and then fixed with very fine sutures over the wound. Supple-

mental treatment is usually with sulfadiazine administered by mouth and intramuscular injections of penicillin. In a series of 10 cases there were nine cures and one death.

PERSKY, Philadelphia.

Miscellaneous

PROPHYLAXIS OF WHOOPING COUGH. MAX J. FOX and REUBEN SNARTEMO, Am. J. Dis. Child. **78**:677 (Nov.) 1949.

Writing from the Department of Internal Medicine, Marquette University School of Medicine, Fox and Snartemo review the literature and discuss the problem of reducing whooping cough incidence in babies newborn or under 6 months of age. The mortality is still high in spite of efforts at vaccination, both of the mother during pregnancy and of the infant soon after birth. Nothing is said in this paper concerning room or cubicle sterilization or administration of "drops" in the nose and throat. Ear, nose, and throat consultation is not mentioned but this is a field for the enthusiastic beginner, especially the bacteriologist. The virus is still unidentified and resists all known methods of destruction. With the new electronic magnification, one may hope for identification, but, even so, "cure" may still be difficult, and prophylaxis must still be the watchword. The authors conclude that whooping cough continues to be a major cause of death in early infancy. Under 6 months of age, immunization is difficult or impossible; after the sixth month, "a high degree of success may be expected." A quotation from C. C. Dauer (Reported Whooping Cough Morbidity and Mortality in the United States, *Pub. Health Rep.* **58**:661 [April 23] 1943) states that "it causes more deaths in children under 2 years of age than any other acute infection with the exception of pneumonia and the diarrheas."

I. W. VOORHEES, New York.

MANDIBULAR GROWTH DISTURBANCE IN RHEUMATOID ARTHRITIS OF CHILDHOOD. MILTON B. ENGEL, JULIUS RICHMOND, and ALLAN G. BRODIE, Am. J. Dis. Child. **78**:728 (Nov.) 1949.

This seemingly thorough study was undertaken in the Departments of Orthodontia and Pediatrics, University of Illinois College of Dentistry and Medicine. Six cases are described in detail, but many others were under observation. The incidence of rheumatoid arthritis in children is estimated to be about 4.9% of the total incidence of this disease. The arthritis may be monoarticular, especially at onset, which is a reversal of the old opinion that no rheumatoid arthritis is ever monoarticular. Involvement of a temporomandibular joint may be the first sign of the disease in children. The reaction to the infection engenders infiltration, fibrosis, and ultimate fixation. A laminagram may make this apparent. Growth of the mandible may be decelerated, resulting in an asymmetry of the right and left sides of the lower jaw. While the soft tissues continue to grow, especially in the floor of the mouth, in disassociation with bony development, encroachment on the pharyngeal area may become serious in its effect on breathing and swallowing. This is essentially a study in pathology, and little is said concerning prophylaxis or therapeusis.

I. W. VOORHEES, New York.

ACUTE RESPIRATORY DISORDERS AMONG NEWBORN INFANTS IN OBSTETRIC NURSERIES: EXPERIENCE IN NEW YORK CITY, 1940 TO 1948. HAROLD ABRAMSON, Am. J. Dis. Child. **78**:883 (Dec.) 1949.

Abramson's article is largely a query into the incidence of respiratory infections in newborn infants. He states that insufficient data are at hand and suggests that the subject be pursued further by clinicians. C. A. Smith in his textbook (Neonatal Immunology, in the Physiology of the Newborn Infant, Springfield, Ill., Charles C Thomas, Publisher, 1945, Chap. 13, pp. 280-297) pointed out that passive immunization of the newborn may be obtained from the mother in some instances. In some outbreaks, the Staphylococcus aureus has been found in the nasopharyngeal secretions of the infant. Postmortem examinations have disclosed capillary bronchitis or bronchopneumonia with multiple abscesses. S. D. Collins (personal communication to the author) thinks that "respiratory infections in minor form occur during the neonatal period with greater frequency than is commonly supposed."

[Abstractor's Comment: All respiratory infections are acquired from some person who is a "carrier" or who is living in the same breathing zone as the unwilling host. Frequent growth of cultures from material from the throats and noses of infants is in order. The hospital staff often carry micro-organisms unwittingly; therefore masking of the nose and face is imperative. Even so, one cannot be certain that germs do not get into the atmosphere. Speaking ex cathedra, an ear, nose, and throat specialist should be in constant attendance, or daily attendance, in every "Babies Ward."]

I. W. VOORHEEES, New York.

INVOLVEMENT OF THE NERVOUS SYSTEM IN INFECTIOUS MONONUCLEOSIS. T. C. BERNSTEIN and H. G. WOLFF, Ann. Int. Med. **33**:1120 (Nov.) 1950.

A review of the literature up to the time of publication of this paper revealed 28 reports, including 46 patients, describing involvement of the nervous system in infectious mononucleosis. The nervous system appears to be affected in less than 1% of patients with infectious mononucleosis.

Many parts of the nervous system may be affected in this disorder, the symptomatology depending on the site of maximal involvement. Serous meningitis, meningitis, encephalitis, meningoencephalitis, polyneuritis, and peripheral neuropathy have been described. It appears that the nervous system is directly invaded by the unknown agent causing the disease.

Clinical evidence of involvement of the nervous system occurs most commonly in males. The changes in the spinal fluid consist of an increase in lymphocytes and/or protein. Changes in the cerebrospinal fluid, as evidence of invasion of the nervous system without any sensory, motor, or mental defects, have been described, and there is suggestive evidence that this is a common occurrence. The prognosis in the series of cases described is excellent, 85% of the patients showing complete recovery.

Since the clinical picture produced by involvement of the nervous system is indistinguishable from that caused by many other agents, the authors advise that heterophil-antibody tests be made on all patients with "idiopathic" diseases of the nervous system. This is particularly important since the systemic signs of infectious mononucleosis may be minimal.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL.].

THE EARLY DIAGNOSIS OF BRONCHOGENIC CARCINOMA. EDWARD A. LOOPER and RICHARD J. CROSS, Ann. Otol. Rhin. & Laryng. **58**:1113 (Dec.) 1949.

Looper and Cross report 29 cases of bronchogenic carcinoma diagnosed by bronchoscopy or exploratory thoracotomy with biopsy of malignant tissue or mediastinal nodes. Bronchoscopy afforded positive material for biopsy or cytological examination or both in 65.5% of the patients.

Of the 28 patients upon whom bronchoscopy was performed, a tentative diagnosis of pulmonary carcinoma was made for 26. The results of the biopsy examination were positive in 7 cases (24%), the results of cytological study of bronchial secretions were positive in 11 cases (55%), and in 5 cases (28%) malignant material was found in the sediment of the bronchial secretions.

It is concluded that bronchial carcinoma should be considered an emergency, and if suspected, an exploratory thoracotomy is advisable, even though bronchoscopic and roentgenographic evidence may be lacking.

STEELE, Kansas City, Kan.

INFECTIOUS MONONUCLEOSIS WITH NEUROLOGIC COMPLICATIONS. VERA B. DOLGOPOL and GEORGE S. HUSSON, Arch. Int. Med. **83**:179 (Feb.) 1949.

Dolgopol and Husson report their observations on a patient who was ill with infectious mononucleosis with severe neurological complications. Until recently, infectious mononucleosis has been regarded as a mild disease, with few complications and with a uniformly favorable outcome. It is certain now that several forms of serious complications are associated with this illness, including hepatitis, thrombocytopenia, myocarditis, spontaneous rupture of the spleen, and involvement of the central and peripheral nervous systems. Multiple serious complications may occur in the same patient.

The patient whose case is described was a 19-year-old girl whose symptoms began with malaise, fatigue, backache, and weakness of the legs. In two days there were photophobia, diplopia, and edema of the eyelids. Within five days after the onset of the symptoms, the deep reflexes were noted to be sluggish, and within seven days there was stiffness of the neck. The cerebrospinal fluid was under increased pressure and contained 50 cells per cubic millimeter, with a normal sugar content and no increase in protein. The heterophil-antibody test gave a positive reaction in a dilution of 1:448. On the following day the Kernig and Brudzinski signs were positive; the patient was unable to move her legs and had difficulty in swallowing. The reaction to the heterophil-antibody test was positive in 1:1,792 dilution. Nine days after the onset of the symptoms the patient died.

Examination of the brain revealed severe degenerative changes in the nuclei of the third and fourth cranial nerves and in the inferior reticular nucleus of the medulla. There was degeneration of the Purkinje cells of the cerebellum. In the spinal cord, hemorrhages were present in the gray matter, mainly in the posterior horns, but in the lumbar region the hemorrhages had spread into the anterior horns as well.

Diverse neurological symptoms in infectious mononucleosis may precede the hematological changes and the appearance of lymphadenopathy and splenomegaly. Such situations invariably present considerable diagnostic difficulties. The manifestations may be similar to those of meningitis, encephalitis, poliomyelitis, or polyradiculoneuritis. Great aid in the diagnosis of this illness can be derived from a test for the heterophil-antibody, as it has been positive in practically all reported cases of infectious mononucleosis with neurological manifestations. It would seem advisable, therefore, to perform that test on all patients with obscure or atypical acute neurological manifestations.

GUTTMAN, Wilkes-Barre, Pa. [A. M. A. ARCH. NEUROL.].

THE PREVENTION OF RESPIRATORY INFECTIONS IN CHILDREN, INCLUDING SULFONAMIDE PROPHYLAXIS. J. KELLER MACK, Illinois M. J. **98**:224 (Oct.) 1950.

In this discussion Mack considers only the complications of the common cold and respiratory infections that are of bacterial origin. In the prevention of repeated respiratory infections, each patient is an individual problem. Dietary or vitamin deficiencies and faulty hygienic measures should be corrected.

Because pharyngeal lymphoid tissue serves a useful purpose, especially in children under 6 years of age, indiscriminate tonsil and adenoid surgery in this age group should be condemned. There is little to be done to raise a child's natural immunity.

During the past three winter seasons 54 children were given sulfonamides daily for the prevention of respiratory infections. Their ages ranged from 2 to 15 years. These children not only fared better than they had previously but had fewer infections than their brothers or sisters and fewer infections than most normal children of their age, with one exception.

BARBOUR, Peoria, Ill. [A. M. A. Am. J. DIS. CHILD.].

AN EXPERIMENTAL PROGRAM FOR HARD OF HEARING CHILDREN. D. E. MORLEY, J. Speech & Hearing Disorders **13**:337 (Dec.) 1948.

Hearing aids and a six weeks' summer program of instruction in acoustic training, lipreading, speech, and correlated activities were provided for a group of hard of hearing children for whom no further medical treatment was indicated and for whom no special class facilities were available. Acoustic examinations were made prior to the program, at the time of selection of the hearing aids, at the conclusion of the training program, and approximately six months later. Tests of achievement, general ability, and personality and social adjustment were made during the program and approximately six months later. The observable results or trends in this project were obtained from a comparison of each child's own responses, deevlopment, or changes as recorded on the first tests and on the retests. Supplementary information was received from the parents of the children.

PALMER, Wichita, Kan. [AM. J. DIS. CHILD.].

A Preliminary Report of a Diagnostic Test for Aphasia. Joe R. Brown and Hildred Schuell, J. Speech & Hearing Disorders **15**:21 (March) 1950.

A diagnostic test for aphasia was developed at the Veterans Administration Hospital in Minneapolis. An attempt was made to avoid being influenced by preconceived ideas of cerebral localization or by standard aphasic categories and to develop an objective outline to reduce the effect of different examiners. The test was designed to sample sufficient and various language components, to be of aid in planning a therapeutic program, and to measure progress of individual patients. The test consists of a series of questions and stimuli grouped under four broad heads: (1) primary associations, concerned with recognition of voice, name, sources of sound, familiar songs, etc.; (2) symbol associations, concerned with inanimate objects, parts of objects, etc.; (3) elaboration of auditory comprehension ("What's your name?"), concerned with simple directions, finding errors, paragraph comprehension, etc., and (4) conceptual processes, concerned with comprehension of prose read aloud, retention, etc.

Some patients have difficulty identifying parts of the body who do not have difficulty identifying objects. Other difficulties seem to be impairments in the ability to find or select specific words voluntarily. It is probable that severe sensory speech defects impair normal function of motor mechanisms and vice versa. No attempts were made in this study to correlate the findings with detailed localizations of the lesions observed by other means.

Palmer, Wichita, Kan.

Muscle Training for Cerebral Palsied Speech Cases. Harold Westlake, J. Speech & Hearing Disorders **16**:103 (June) 1951.

Careful attention should be given to the use of motor training in speech therapy in cases of cerebral palsy. A detailed scrutiny of muscle activity and patterns of movement frequently reveal remediable defects which interfere with speaking efficiency. Systematic evaluation and muscle training techniques offer a great deal to the speech therapist.

Palmer, Wichita, Kan.

An Exploratory Investigation of the Self-Concept of Stutterers. Fred F. Fiedler and Joseph M. Wepman, J. Speech & Hearing Disorders **16**:110 (June) 1951.

Ten men stutterers were compared with six nonstutterers by means of the Q-technique. On the basis of 76 statements descriptive of personality traits, self-concepts of stutterers in this study showed no characteristic differences from those of the nonstutterers.

Palmer, Wichita, Kan.

Suprahyoid Neck Infections. Colby Hall, Laryngoscope **60**:779 (Aug.) 1949.

Hall discusses certain suprahyoid neck infections: pharyngomaxillary, masticator, and Ludwig's infections. There is a brief anatomical description with illustrations, followed by the signs and symptoms to be expected for each. Treatment is described and cases are reported.

Hitschler, Philadelphia.

Plastic and Reconstructive Surgery. Sanford R. Dietrich and James Barrett Brown, Laryngoscope **60**:1129 (Nov.) 1950.

This paper consists of a comprehensive review of reconstructive and plastic surgery. Operations performed on other parts of the body in addition to the nose and the ear are included. There is a large bibliography.

Hitschler, Philadelphia.

Respiratory Complications Associated with Demerol. Thomas F. McDermott and E. M. Papper, New York J. Med. **50**:1721 (July 15) 1950.

McDermott and Papper's observations were made in the Department of Anesthesia at Columbia-Presbyterian Medical Center. Meperidine (demerol®) has been generally accepted as an insignificant respiratory depressant and as having an antispasmodic effect similar to

atropine. This is an understatement, say these investigators, for respiratory depression has been noted, especially in debilitated persons and in those having cerebral arteriosclerosis. Two cases of maternal cyanosis and two deaths from overdose, one with suicidal intent, have been reported. Meperidine is a drug of unquestioned clinical value but cannot be used with impunity. When it was given intravenously with thiopental sodium (pentothal®)-nitrous oxide anesthesia, bronchospasm was observed in three cases. The mechanism is not understood but may be synergistic. The authors state, "The concept that Demerol is not depressant to respiration and has no adverse effects upon breathing patterns cannot be accepted in all circumstances."

I. W. Voorhees, New York.

Traction Diverticulum of the Thoracic Esophagus Caused by Suppurative Lung Abscess. Walter E. Bugden, New York J. Med. **50**:2568 (Nov. 1) 1950.

A Polish woman, aged 50, was admitted to Syracuse Memorial Hospital on Sept. 11, 1947. Previously, elsewhere, a right lower lobe abscess had been opened and drained incompletely. The sputum was green, nonodorous, and in amounts of from ½ to 3 oz. (14.2 to 85.1 gm.) daily. Diagnosis was made definite by bronchogram. The operation, on September 17, disclosed a hard, massive, infected right lower lobe. Ligation and dissection were carried out, working from the posterior chest wall where rib resection had been done previously. A chronic lung abscess was found. The esophagus was found attached to the posterior facet of the lower lobe and was pulled upward by adhesions out of the mediastinum. A traction diverticulum 2 by 4 cm. was firmly embedded in the mass. This was opened and resected and the esophagus closed with two layers of fine nonabsorbable surgical suture. The muscle was so attenuated that it was questionable whether the sutures would hold. Vein, artery, and bronchus were ligated and divided en masse because separation of each from the other was impossible. Subsequent healing was complete in four weeks, in spite of a small fistula which intervened but was successfully dealt with. Three years later there were neither pulmonary nor esophageal symptoms.

I. W. Voorhees, New York.

Biochemical Aids in the Diagnosis of Cancer. Oscar Bodansky, New York J. Med. **50**:2789 (Dec. 1) 1950.

Bodansky writes from the Memorial Hospital and the Sloan-Kettering Institute for Cancer Research and states that for more than 50 years in a search for a diagnostic tool in cancer there has been alternation between hope and disappointment. From 1940 to 1948, 60 procedures were tried. The latest seems to be that of Huggins, Miller, and Jensen, who experimented with coagulation of serum proteins according to a formula containing iodoacetate as an index. Bodansky and collaborators repeated these experiments, but noted considerable discrepancy in results. Infection or operation seemed to vitiate the metabolism of protein. Both corticotropin and cortisone were used in this research with varying reactions. Specific types of cancer were investigated: cancer of the liver, cancer of the ducts and acini of the pancreas, tumors of islet cells with hyperinsulinism, and cancer of the adrenals. In brief, biochemical aids in diagnosis of cancer may be general or specific, but a general test has not yet been established which will bear scientific scrutiny. Investigation of cancer in the second group of specific organs offered more information, since we know something of the biochemistry of such organs as the prostate, adrenals, and pancreas. However, in every instance we must go back to the general history, complete physical examination, and all aids to be derived from laboratory tests; and, if these do not completely determine an accurate diagnosis, they do help in management of the future daily life of the patient. There are 27 references to the literature.

I. W. Voorhees, New York.

A Program for the Management of Hare Lip and Cleft Palate. Charles George Neumann, New York J. Med. **50**:2952 (Dec. 15) 1950.

In cleft palate work, Neumann states that the combined services of general practitioner, pediatrician, orthodontist, speech therapist, psychiatrist, vocational counselor, anesthesiologist, and plastic surgeon are required. The age for harelip repair is from 1 day to 6 weeks. The feeding and general physical condition of the infant must be studied. From the fourth to the

tenth day the time is less propitious for surgery, and if weight loss (the infant weighs under 7 lb. [3,175 gm.]) or physiological jaundice is present surgery is "undesirable." Dropper feedings slowly given are recommended. General anesthesia is followed by vomiting, therefore local anesthesia (nerve block) is preferable. Harelip calls for a good cosmetic result, cleft palate for functional adequacy. The operation for the latter is carried out at 1½ to 2 years. Aspiration pneumonia or middle ear infections may be troublesome. Residual speech defects must be overcome by a special teacher. Unilateral nasal deformity goes with harelip defect. This should be dealt with in early adult life. Psychiatric aspects concern both patient and parents. Subsequent children are not likely to have these defects. This may seem highly important to all concerned. At present, the etiology is not sufficiently known to offer likelihood of prevention of these deformities.

I. W. VOORHEES, New York.

THE RECOGNITION AND TREATMENT OF INCREASED INTRACRANIAL PRESSURE IN INFANCY AND CHILDHOOD. BARNES WOODHALL, Pediatrics **2**:533 (Nov.) 1948.

Recognition of increased intracranial pressure in infants and children is directly influenced by two anatomic factors: 1. The skull is an expansible structure, with open fontanels and infused suture lines. 2. The normal increment of brain volume is trebled by 3 years of age and attains adult size only at 15 years. The roentgenogram of the skull of the growing child, therefore, differs from that of the adult and may reveal the imprint of the expanding hemisphere on its inner surface. Roentgenologic diagnosis must consider these variations in normal growth and is dependent on the demonstration of an abnormally large skull, convolutional atrophy, separation of suture lines, and erosion of the sella turcica. Pressure of the normally growing brain on the skull is graphically demonstrated by roentgenogram in the clinical syndrome of craniosynostosis; pathological alterations are best seen in advanced instances of brain tumor.

Study of roentgenograms of 100 consecutive patients with brain tumors showed that the presence of increased intracranial pressure could be suspected in 63. A major portion of the group presented a short and almost fulminating onset of symptoms, with about half complaining of symptoms of increased intracranial pressure (headache, vomiting, and visual difficulty) and the remainder complaining of symptoms suggesting a specific neurological defect (staggering gait and muscle weakness). In the neurological examination of these 100 patients, 88 showed increased intracranial pressure through ophthalmoscopic examination alone. Other pathological evidence included the presence of an enlarged head, a cracked-pot sound on percussion of the skull, and cervical rigidity.

The prognosis for children of this age group with typical histories of tumors is favorable only in the group with cerebellar astrocytomas. The safety of surgical intervention in tumors in children of this age group has been greatly enhanced by technical advances; ignorance of the fundamental tumor process remains profound. The neurosurgeon is dependent on prompt and good pediatric diagnosis.

FROM THE AUTHOR'S SUMMARY [A. M. A. Am. J. DIS. CHILD.].

ERB'S PALSY. B. WOLMAN, Arch. Dis. Childhood **23**:131 (June) 1948.

The end-results of 37 cases of laceration of the peripheral nerve at birth (Erb's palsy) were investigated. The condition is slightly commoner in boys. Injury at birth to the supraclavicular portion of the brachial plexus is the accepted cause of the condition, and, therefore, skilled obstetric technique in delivery, particularly in the use of forceps, is the main factor in avoiding this paralysis.

Large babies are more likely to be injured. A large child in a primigravida needs special care with the delivery of the head and arms. Treatment must be carried out from birth. Children treated within the first month of life do uniformly well and results are 100% successful. If treatment is delayed, contractures causing impaired function of the shoulder and elbow will develop quickly.

KELLY, Milwaukee [A. M. A. Am. J. DIS. CHILD.].

RECURRENT MIXED TUMOR OF THE CHEEK IN A GIRL AGED 1½ YEARS. M. M. KINAWI and S. M. AIDAROS, J. Roy. Egyptian M. A. **32**:69 (Jan.) 1949.

The authors report a case of mixed tumor of the parotid gland which first occurred when the patient was 2 months old. The child was operated on at 7 months. The tumor recurred

after four months and was removed when the child was 18 months old. The authors have been unable to find a similar case reported for infants or children.

McGuigan, Evanston, Ill. [A. M. A. Am. J. Dis. Child.].

Adenoma of the Bronchi. Eelco Huizinga and J. Iwelma, Ann. oto-laryng. **67**:743 (Nov.-Dec.) 1950.

Seventeen patients with bronchial adenomas and three with tracheal tumors, two of which were cylindromas and one of which was an adenoma, were treated in Groningen, Netherlands. The majority of the adenomas had a typical microscopic appearance, and several of the characteristics are discussed, as some portions of the tumors simulated a cylindroma.

Two main characteristics are discussed at greater length: 1. The classification of the adenoma among tumors is discussed. Huizinga and Iwelma are of the opinion that this benign epithelial tumor is a characteristic localization of the mixed parabuccal tumor, arising from the bronchial mucous glands. Various findings which support this belief are mentioned. 2. No definite rule can be established as to therapy. Several factors should be considered in each case, as, for instance, the age of the patient, the location of the tumor, and the microscopic appearance. Gratifying results have been obtained both by bronchoscopic therapy and by surgery, as the case warranted.

Viole, Los Angeles.

Metastatic Brain Abscess. J. LeBeau and M. Rosier, Rev. neurol. **81**:343 (May) 1949.

LeBeau and Rosier report the results of operation in 18 cases of metastatic cerebral abscess since 1944, when treatment by the method of total ablation and instillation of penicillin was adopted. They conclude that abscess is to be suspected when localizing neurological signs or intracranial hypertension appears during the course of a subacute infection, particularly of the lung. Acute aseptic meningitis, or meningitis that does not clear quickly with penicillin, leads to the suspicion of abscess. Most abscesses in their series appeared in the posterior half of the cerebrum. The abscess is found via a burr hole. If it is acute, it is drained by puncture, and 20,000 to 50,000 units of penicillin is injected. However, if unequivocal improvement does not appear in 24 hours, a flap is turned and the abscess is ablated. In case of chronic or subacute abscess, immediate ablation is carried out. Penicillin treatment is continued for a few weeks afterward.

Ventriculographic examination is not made during the diagnostic stage.

The mortality by this method was 33%, representing a considerable improvement over former results.

The authors feel that the incidence of neurological sequelae is less than with drainage. They carry out the ablation by opening the abscess widely and removing the capsule from the inner surfaces, a procedure which they feel can be carried out with impunity provided removal of the wall and infective tissue is complete.

Legault, Washington, D. C. [A. M. A. Arch. Neurol.].

Treatment of Asthmatic Diseases in Infants and Children with Chamazulen. Sándor Blaszó, Schweiz. med. Wchnschr. **79**: 222 (March 12) 1949.

Chamazulen is a dark blue hydrocarbon, a distillation product of the ethereal oils of Achillea millefolium and Matricaria chamomilla. Its constitution is not fully established. It has been stated that its specific action consists in the liberation of histamine over a protracted period of time, with a consequent diminution of histamine sensitivity. The drug was given intramuscularly in 0.5 to 2% oily solution. The age of the patients varied from 3 months to 9 years. The asthma or spastic bronchitis yielded in all cases to from four to eight injections, of 0.01 to 0.02 gm. each, given at two day intervals; one or two further injections seemed to stabilize the cure. In only three cases was it necessary to give 10, 11, or 12 injections. Occasionally the symptoms became worse, never for more than 56 hours. It is too early to decide whether or not recurrences may happen.

Amberg, Rochester, Minn. [A. M. A. Am. J. Dis. Child.].

PROPHYLAXIS BY PENICILLIN FOR CARRIERS OF THE DIPHTHERIA BACILLUS. C. TORRICELLI and A. MONTAGNA, Lattante **18**:474 (Aug.) 1947.

Twenty-two children, carriers of Corynebacterium diphtheriae, were treated with local applications of penicillin (1,000 units per cubic centimeter) four to six times daily; 4 to 15 days later cultures were negative for the bacilli.

In vitro, 1 unit of penicillin per cubic centimeter was bacteriocidal and higher dilutions were bacteriostatic for diphtheria bacilli. In no instance did the penicillin change a toxin-producing diphtheria bacillus to a nontoxin-producing bacillus. Most of the strains of C. diphtheriae found in the carriers were not toxic to guinea pigs.

HIGGINS, Boston [AM. J. DIS. CHILD.].

A CONTRIBUTION TO THE STUDY OF OTOGENIC CEREBELLAR ABSCESSES. P. ZDRAVEV, Ann. oto.-laryng. **67**:782 (Nov.-Dec.) 1950.

1. In the treatment of cerebellar abscesses, the bony focus must be eliminated with extensive exposure of the dura mater.

2. Chemotherapeutic and antibiotic agents must be applied locally as well as administered systemically. Lumbar and suboccipital routes are of great importance, as they are the most effective against meningitis and encephalitis. These routes should be used as long as the intramuscular route is used, even in the absence of meningitis.

3. A small incision in the abscess is essential for observation and aspiration of its contents.

4. Packing of the abscess should be avoided as much as possible.

5. Drains should be eliminated.

6. Systemic treatment as well as blood transfusions should be resorted to as indicated.

7. Complete extirpation of the otogenic cerebellar abscess is a hazardous procedure, particularly if the abscess is large, as too much healthy tissue is sacrificed.

8. By the above procedure the duration of treatment is greatly shortened. Zdravev does not wish to convey the impression that all cerebellar abscesses will be cured by the methods outlined above.

VIOLE, Los Angeles.

NYSTAGMUS OF THE SOFT PALATE DUE TO TRAUMA. M. KLINGLER, Schweiz. Arch. Neurol. u. Psychiat. **64**:253, 1949.

A man aged 35 sustained a severe injury to his head. Examination a month later revealed pronounced nystagmus in all directions of gaze, Horner's syndrome, and hemiataxia of the cerebellar type on the left side with hemianesthesia for pain and temperature on the right. A year after the accident, nystagmus was present only on upward gaze, and impairment of sensation for pain and temperature was limited to the ulnar portions of the right hand and forearm. On the occasion of the second examination, however, rhythmic myoclonic twitching involving both sides of the soft palate was observed. Similar rhythmic movements were also seen in the posterior wall of the pharynx and the left vocal cord.

In four cases collected from the literature palatal nystagmus was attributed to trauma, but in only two of these was the evidence for a traumatic origin of the condition particularly convincing. One of the two cases was similar to Klinger's case in that myoclonus of the right halves of the soft palate, pharynx, and larynx was associated with hemiataxia on the same side and with hemianesthesia for pain and temperature on the left.

DANIELS, Denver [A. M. A. ARCH. NEUROL.].

Special Reports

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

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Cleartone Regency Model

Mfr., American Sound Products, Inc.
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Dahlberg Junior Model D-2

Mfr., The Dahlberg Company
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Minneapolis 16

Dysonic Model 1

Mfr., Dynamic Hearing Aids, Inc.
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Brooklyn 16

Electroear Model C

Mfr., American Earphone Co., Inc.
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New York 17

Gem Model V-35

Gem Model V-60

Mfr., The Gem Ear Phone Co., Inc.
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Maico Atomeer

Maico UE Atomeer

Maico Quiet Ear Models G and H

Maico Model J

Mfr., The Maico Company, Inc.
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Mfr., Mears Radio Hearing Device Corp.
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Micronic Star Model

Mfr., Micronic Company
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Microtone Classic Model T-9

Microtone Model T-10

Microtone Model T612

Microtone Model 45

Mfr., The Microtone Company
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National Cub Model (C)

National Model D (Duplex)

National Standard Model (T)

National Star Model (S)

National Ultrathin Model 504

National Vanity Model 506

Mfr., National Hearing Aid Laboratories
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Los Angeles 14

Otarion Model E-4

Otarion Models F-1, F-2, and F-3

Otarion Model G-2

Mfr., Otarion, Inc.
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Radioear All-Magnetic Model 55

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Mfr., National Hearing Aid Laboratories
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Silvertone Model J-92

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925 S. Homan Ave.
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Silvertone Model P-15

Mfr., W. E. Johnston Mfg. Co.
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Minneapolis 8
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Chicago 7

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Mfr., Solo-Pak Electronics Corp.
Linden St.
Reading, Mass.

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Sonotone Model 900

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Telex Model 22

Telex Model 97

Telex Model 99

Telex Model 200

Telex Model 300-B

Telex Model 400

Telex Model 1700

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Mfr., Tonamic, Inc.
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Directory of Otolaryngological Societies *

INTERNATIONAL

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY

Meeting: Third Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology
Place: Habana, Cuba. Time: January 20-24, 1952.
President: Dr. José Gros.
Secretary: Dr. Pedro Hermandez Gonzalo, Calle 8, 358, Habana, Cuba.

THIRD LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY

Place: Caracas, Venezuela. Time: 1953.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. Carl H. McCaskey, 20 N. Meridian St., Indianapolis.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Place: Chicago. Time: June 11-13, 1952.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Derrick Vail, 700 N. Michigan Avenue, Chicago 11.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago. Time: Oct. 14-20, 1951.

AMERICAN BOARD OF OTOLARYNGOLOGY

Secretary: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Palmer House, Chicago. Time: Oct. 9-12, 1951.
Place: Royal York Hotel, Toronto, Canada. Time: May 13-16, 1952.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

President: Dr. Herman J. Moersch, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia 2.
Place: Royal York Hotel, Toronto, Canada. Time: May 23-24, 1952.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Royal York Hotel, Toronto, Canada. Time: May 20-22, 1952.

SECTIONS:

Eastern.—Chairman: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Place: Waldorf-Astoria, New York. Time: Jan. 11, 1952.
Southern.—Chairman: Dr. Lester A. Brown, 490 Peachtree St., Atlanta.
Place: Academy of Medicine, Atlanta. Time: Jan. 14, 1952.
Middle.—Chairman: Dr. Ralph J. McQuiston, 20 N. Meridian St., Indianapolis.
Place: Indianapolis Athletic Club, Indianapolis. Time: Jan. 28, 1952.
Western.—Chairman: Dr. Howard P. House, 1136 W. 6th St., Los Angeles.
Place: County Medical Building, Los Angeles. Time: Jan. 19-20, 1952.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse, N. Y.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago.
Place: Royal York Hotel, Toronto, Canada. Time: May 18-19, 1952.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

President: Dr. Norman N. Smith, 291 Whitney Ave., New Haven 11, Conn.
Secretary: Dr. Joseph G. Gilbert, 111 E. 61st St., New York 21.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. William H. Evans, 24 Wick Ave., Youngstown 3, Ohio.
Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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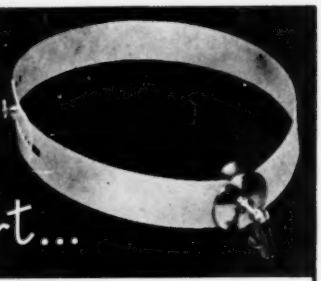

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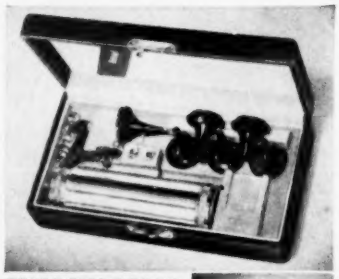

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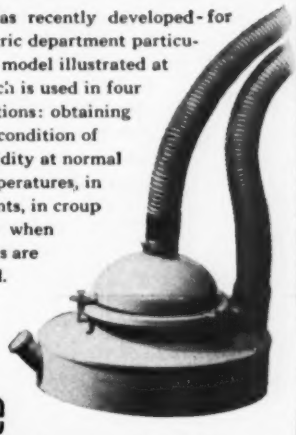

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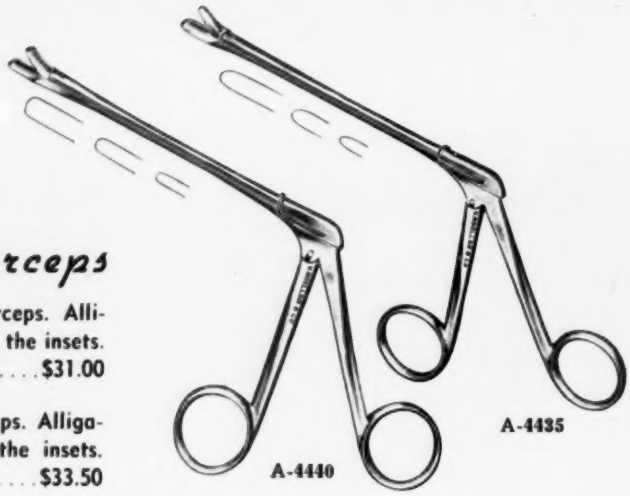

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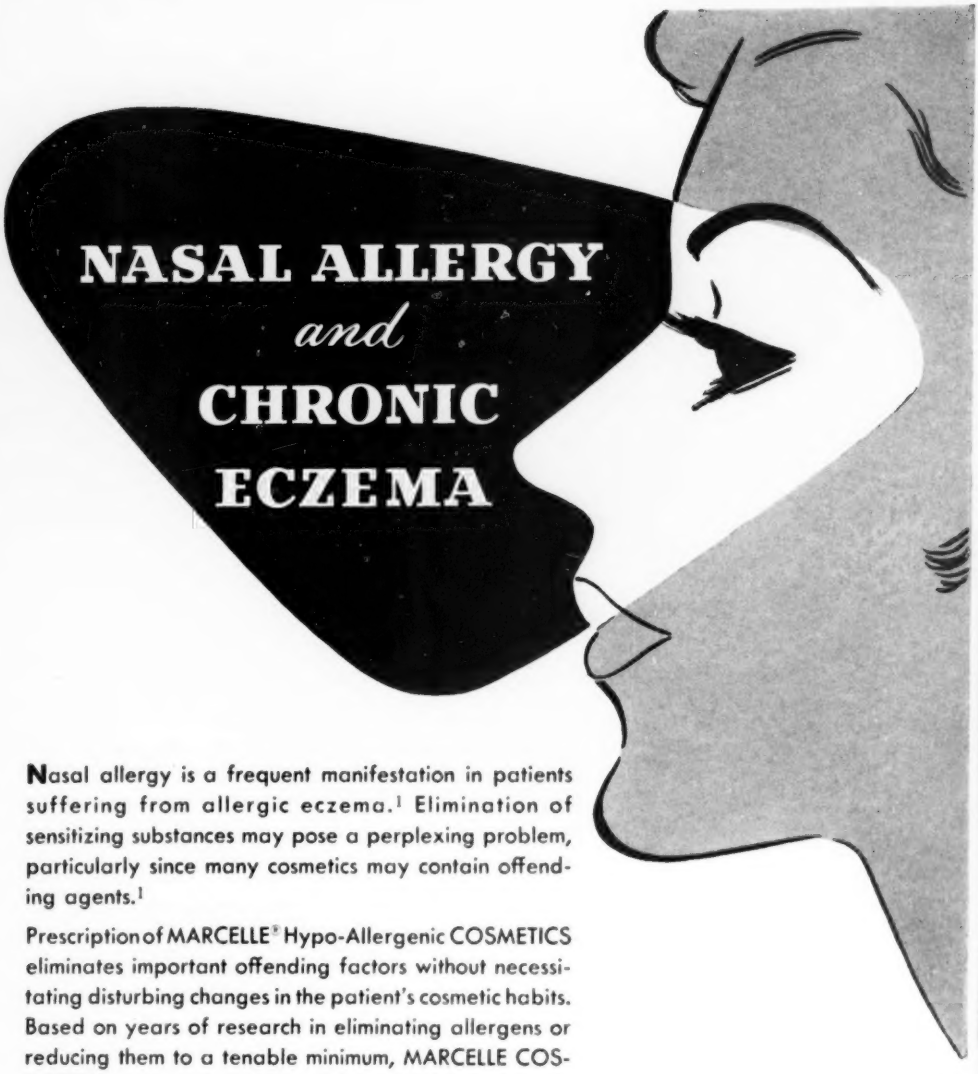

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1. Waldbott, G. L.: Management of Allergic Eczema. Am. Pract. 4:59-63 (Oct.), 1949.

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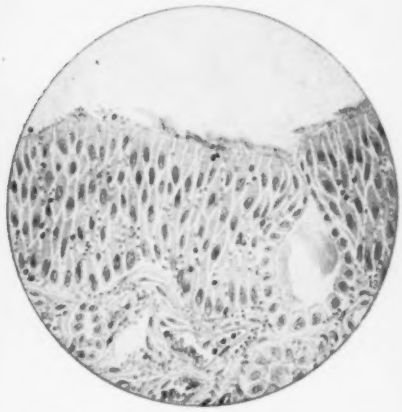

Nasal membrane showing increased leukocytes with denudation of cilia.

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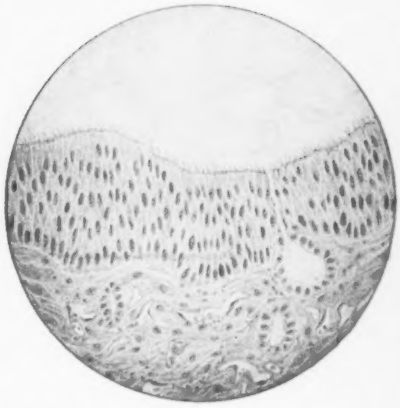

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